THE PREVALENCE OF CORONARY RISK FACTORS AMONG CHILDREN, AGES 11 TO 13, IN SELECTED WESTERN CAPE SCHOOLS

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DECLARATION

I, the undersigned, hereby declare that the work contained in this thesis is my own original work and has not previously in its entirety or in part been submitted at any university for a degree.

Signature Date

ABSTRACT (ENGLISH)

Numerous studies have shown that coronary artery disease (CAD) has its origin in childhood. Several risk factors that increase a person's risk for the development of CAD are prevalent amongst children. South African statistics concerning the prevalence of these risk factors are limited.

Research has shown that early intervention to eliminate risk factors can decrease the risk for the development of CAD.

The purpose of this study was to determine the prevalence of certain coronary risk factors amongst children aged 11 to 13 years in certain Western Cape schools. Certain selected factors were tested. These included obesity, lack of physical activity, hypertension, low physical fitness (VO_2max), a family history associated with an increased risk, exposure to cigarette smoke, prevalence of diabetes mellitus and an unhealthy diet.

The sample consisted out of 288 children and was made up by 154 boys and 134 girls. Certain anthropometrical measurements (stature, weight, skinfolds, waist and hip circumferences) were taken. Activity levels, family history, exposure to cigarette smoke, prevalence of diabetes mellitus and diet, were measured by means of questionnaires. Physical fitness (VO_2max) was tested with a three-minute step-test. A sphygmomanometer was used to measure blood pressure. Depending on the circumference of the child's arm, a paediatric or adult size cuff was used.

The results of the study showed that 22.01% of the boys and 59.7% of the girls had a percentage body fat so high that it was considered a coronary risk factor. Physical fitness levels were considered risk factors in 2.6% of the boys and 9% of the girls. A very high percentage of the children tested had a family history associated with an increased risk for the development of CAD (73.38% of the boys and 78.36% of the girls). Systolic hypertension was prevalent among 22.01% of the boys and 23.13% of the girls.

Diastolic hypertension was only prevalent among 5.19% of the boys and 5.97% of the girls. Low activity levels were considered a risk factor in 31.17% of the boys and 39.55% of the girls. Out of all the subjects, 32.47% of the boys and 37.31% of the girls were exposed to cigarette smoke on a daily basis.

The results of this study shows that certain coronary risk factors are quite common amongst children. Prevention programmes that focuses on elimination of coronary risk factors, such as hypertension, inactivity and obesity, is essential for the prevention of subsequent coronary artery disease in adults.

OPSOMMING (AFRIKAANS)

Verskeie studies het al bewys dat koronêre hartvatsiekte (KHS) reeds sy ontstaan het in kinders van baie jong ouderdomme. Verskeie risikofaktore wat tot die latere ontstaan van KHS lei kom ook onder jong kinders voor. Statistiek ten opsigte van die voorkoms van hierdie risikofaktore onder kinders in Suid-Afrika is egter baie beperk.

Verskeie navorsing toon dat vroeë intervensie kan lei tot 'n verlaging in risiko vir die ontwikkeling van KHS op 'n latere stadium.

Die doel van die studie was om die voorkoms van sekere koronêre risikofaktore in kinders in Suid-Afrika te ondersoek. Sekere risikofaktore is ondersoek, dit het ingesluit, obesiteit, lae fisieke aktiwitietsvlakke, hipertensie, lae fisieke fiksheid (VO_2maks), 'n familie geskiedenis wat geassosieer word met 'n verhoogte risiko, blootstelling aan sigaret rook, die voorkoms van diabetes mellitus en 'n swak dieet.

Die steekproef het bestaan uit 288 kinders waarvan 134 meisies en 154 seuns was. Verskeie antropometriese meetings (lengte, massa, velvoue, middel- en heup omtrekmates) is geneem. Aktiwiteitsvlakke, familiegeskiedenis, blootstelling aan sigarette rook, voorkoms van diabetes mellitus en dieet is deur middel van vraelyste vasgestel. Fisieke fiksheid (VO_2maks) is deur middel van 'n drie-minuut-opstaptoets vasgestel. Bloeddruk is met 'n sfigmomanometer gemeet. Afhangend van die omtrek van die kind se arm is 'n pediatries- of volwasse-grootte drukband gebruik.

Persentasie liggaamsvet was by 22.01% van die seuns en 59.7% van die meisies so hoog dat dit as 'n risikofaktor beskou kan word. Fisieke fiksheidsvlakke kan by 2.6% van die seuns en 9% van die meisies as 'n risikofaktor beskou word. 'n Baie hoë persentasie van die kinders het 'n familiegeskiedenis gehad wat geassosieer word met 'n verhoogde risiko vir die ontwikkeling van KHS (73.38% van die seuns en 78.36% van die meisies). Sistoliese hipertensie het onder 22.01% van die seuns en 23.13% van die meisies voorgekom. Diastoliese hipertensie het baie minder voorgekom as sistoliese hipertensie (5.19% van die seuns en 5.97% van die meisies). Lae aktiwiteitsvlakke het

onder 31.17% van die seuns en 39.55% van die meisies voorgekom. 'n Redelike hoë persentasie van die kinders word daagliks aan sigaretrook van hul ouers of oppassers blootgestel (32.47% van die seuns en 37.31% van die meisies.)

Die resultate van die studie dui aan dat daar 'n redelike hoë voorkoms van sekere koronêre risikofaktore onder kinders is. Ondersoek moet ingestel word na moontlike, goed gestruktureerde intervensieprogramme.

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CHAPTER I INTRODUCTION

Coronary artery disease (CAD) is fast becoming the leading cause of morbidity and mortality, not just in South Africa, but all over the world as well. It has been proved that risk factors for CAD at a young age, although clinical symptoms may only manifest at a later stage (Raitakari *et al.*, 1997). The World Health Organisation (WHO) predicted that in the year 2020 CAD would be one of the leading causes of death in developing countries (Steyn *et al.*, 2000). According to the American College of Sports Medicine (Roitman *et al.*, 1998:225) CAD is the leading cause of death in industrialised countries.

CAD was recognised as a disease in the 19th century, but it was only in the 20th century that it got the attention needed (Roitman *et al.*, 1998:225). The first studies on CAD, its causes and risk factors, were all done on white adult men. The urgency for intervention seemed the greatest amongst this group (Keys, 1970:2). Figure 1.1 illustrates the percentage of CAD related deaths amongst white men in the United States (U.S.) in 1962.

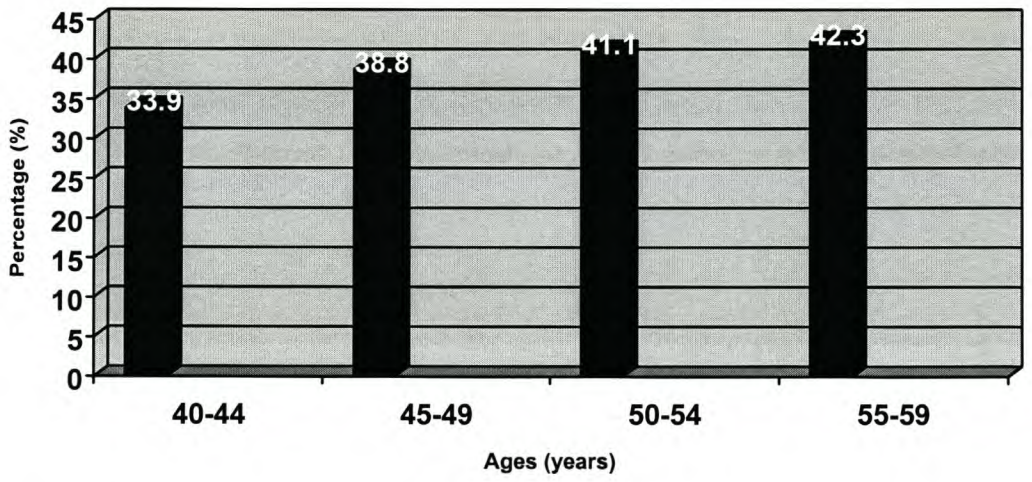

Figure 1.1 Percentage CAD related deaths amongst white U.S. males in 1962 (Adapted from Keys, 1970:2).

As CAD became one of the major causes of death, research intensified and studies such as the ones done in Minnesota (1947-1948), Framingham (1948-1950) and later New York provided more insight into the causes and associated risk factors (Keys, 1970:1; Rossouw, 1990:1).

Holman (1961) was first to regard CAD as a paediatric problem. However, major studies on children only began in the 1970's (Strong, 1983).

More and more research shows that coronary artery disease starts developing in early childhood (Baranowski *et al.*, 1992; McArdle *et al.*, 1996:649; Boreham *et al.*, 1997; Roitman *et al.*, 1998:226; Rolfes *et al.*, 1998:268; Washington, 1999). According to Washington (1999) intervention through risk factor modification can be effective in childhood. The lack of longitudinal studies, according to this author, prevents one to assume in all cases that risk factor modification will reduce morbidity and mortality caused by CAD. It is noted, however, that risk factor modification in childhood has a positive impact on the health and well being of children.

The cost of CAD in the U.S. was estimated in 1994 to be $128 million; this includes medical care and loss of productivity (Plowman and Smith, 1997). If CAD risk factors are detected early, intervention is still possible and positive health practices can be established. This could have a direct impact on the lowering of CAD morbidity and dissuade rising health care costs (Schmidt *et al.*, 1998).

The literature clearly shows that CAD in later life can, in many cases, be attributed to risk factors present in childhood (Baranowski, 1992; McArdle *et al.*, 1996:649; Boreham *et al.*, 1997; Roitman *et al.*, 1998:226; Rolfes *et al.*, 1998:268; Washington, 1999). However, there is little South African literature available on this. If childhood CAD risk factors are the cause of some of the CAD-related deaths in South Africa, the study of the prevalence of these risk factors in children could lead to the decrease in CAD related deaths later in life, if the necessary intervention takes place.

The literature shows that the prevalence of risk factors amongst children is evident in most parts of the world. According to McGinnis (1995) the prevalence of obesity in children is on the rise in the United States. Less than 50% of their children engage in routine physical activity and adding to that, less than 36% of schools offer physical education classes. Sedentary activity has increased, with television watching averaging 24 hours a week. Forty five percent of children snack on high fat, calorie-dense foods at least twice a day.

The objective of this study was to determine what the prevalence of certain CAD risk factors amongst children in the Western Cape, South Africa is. At the same time, hopefully, this study will contribute to the development of structured CAD prevention programmes, especially in children, if the results indicate that to be necessary.

The availability of resources, finances and time determined which risk factors were to be focused on for this study. According to Kenney and Humphrey (1995:18) the American College of Sports Medicine identified eight primary risk factors for CAD; they are age, family history, cigarette smoking, hypertension, hypercholesterolemia, diabetes mellitus, sedentary lifestyle or inactivity and obesity. Other resources add gender to this list (McArdle *et al.*, 1996:648; Roitman *et al.*, 1998:225).

CHAPTER II LITERATURE REVIEW

Numerous studies have found that coronary risk factors are prevalent amongst children. Most of these studies, however, were done in countries other than South Africa. Very few statistics are available on the prevalence of coronary risk factors in South African children. According to Statistics South Africa, children under the age of eighteen years comprise almost half of the South African population (Anon, 1999). Statistics on causes of death in South Africa for 1995 are presented in table 2.1.

Table 2.1 Cause of death statistics in South Africa for 1995 (Adapted from: World Health Organisation, 1996).

Cause of death	Sex	All ages	Ages									
			<1	1-4	5-14	15-24	25-34	35-44	45-54	55-64	65-74	75+
Diseases of the circulatory system	M	19854	7	29	61	191	543	1537	3055	4267	4977	5187
	F	21045	8	14	78	239	600	1135	2093	3562	5106	8210
Hypertensive disease	M	1574	-	-	-	7	34	140	240	339	390	424
	F	2668	-	-	1	10	49	126	275	527	669	1011
Acute myocardial infarction	M	2590	-	-	-	5	40	176	429	627	707	606
	F	1729	-	-	-	2	17	54	144	261	463	788
Other ischaemic heart diseases	M	3459	-	-	-	3	39	218	537	854	912	896
	F	2358	-	-	-	2	16	72	198	393	627	1050
Other forms of heart disease	M	4459	7	28	29	90	206	427	688	860	1049	1075
	F	4700	8	12	49	131	278	334	494	798	1023	1573
Atherosclerosis	M	163	-	-	-	-	1	2	7	15	47	91
	F	229	-	-	-	-	-	1	-	8	29	191

When observing the table above (table 2.1) it is clear that the number of deaths related to atherosclerosis is slightly higher in women than in men, with a definite increase with age.

Basic anatomy of the arteries

Arteries are lined with a layer of active cells, called the endothelium. It controls the passage of substances from the blood to the arterial wall. Underneath the endothelium is the tunica intima. The tunica intima consists out of a thin layer connective tissue cells with an occasional smooth muscle cell. The next layer beneath the tunica intima is the tunica media, which is located between the internal and external elastic laminas. In addition to the elastic connective tissue, the tunica media contains most of the smooth muscles of the arterial wall (Roitman *et al.*, 1998:225-226).

Coronary artery disease

Arteriosclerosis is the term that is used to describe the natural anatomic changes to the arteries caused by aging. These changes include the lost of elastic connective tissue, the increase in diameter of the arteries and the thickening of the intima. Atherosclerosis, on the other hand, is a pathologic phenomenon causing obstructive lesions in the arteries. Normally the endothelium prevents the development of atherosclerosis. When the endothelium is not intact, though, it plays a critical role in the development of the disease. Atherosclerotic lesions forms on the intima of the arteries. Advanced atherosclerosis is characterised by the abnormal growth of the smooth muscle cells of the media (McArdle *et al.*, 1996:648; Roitman *et al.*, 1998:225).

Atherosclerosis begins to develop in the event of an injury caused by substances in the blood or derangement of the endothelium. This causes an inflammatory response that causes the arterial wall to thicken, causing obstruction of blood flow. Tobacco smoke or other chemicals from tobacco, hypertension, hypercholesterolemia or diabetes mellitus can cause injury to the endothelium. Platelets that respond to the injury form small blood clots. The platelets also release a vasoconstrictor that can cause further vascular damage. The hyperthrombotic state causes atherosclerosis to develop. The lesions take the form of fatty streaks, which are the first symptoms of atherosclerosis. As damage and abnormal growth continues the fatty streaks become lipid-filled plaques and fibrous scare tissue. This degenerative process causes the lining of the arteries to roughen and harden, forming plaque. These changes progressively reduce the blood

flow and as a result, the slow flowing blood begins to clot, resulting in a coronary event (figure 2.1) (McArdle *et al.*, 1996:648; Roitman *et al.*, 1998:226).

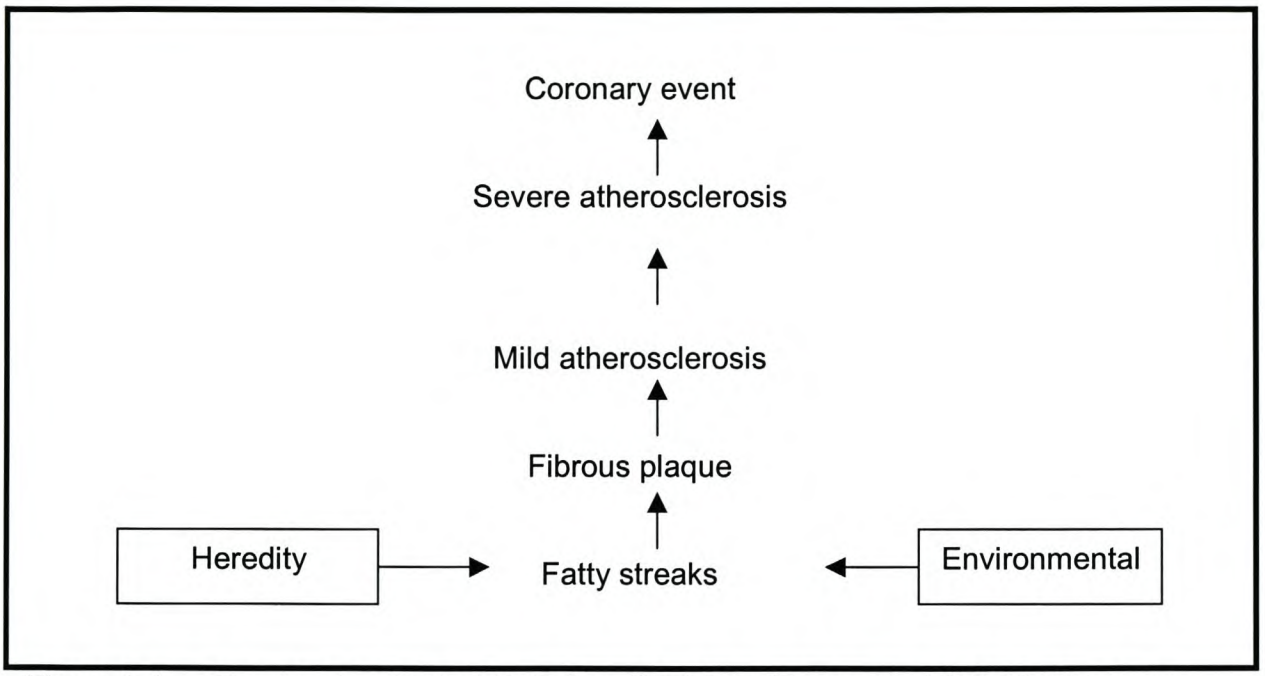

Figure 2.1 The development of CAD (modified from Rossouw, 1990:22; Thompson and Wilson, 1992:1.5).

A hyperthrombotic state may be the result of a genetic trait, or an excessive blood catecholamine concentration. An excessive blood catecholamine concentration may be the result of smoking or psychological stress. Smoking, a sedentary life-style, or elevated low-density lipoprotein (LDL) levels may cause an impaired ability to dissolve intra-arterial thrombi (McArdle *et al.*, 1996:649; Roitman *et al.*, 1998:226).

Evidence of CAD is present in most people. The process probably begins in childhood and progresses over many years, because fatty streaks in the coronary arteries of children are common even at five years of age. Only when 50-70% of an artery occluded, can it be clinically detected (McArdle *et al.*, 1996:649; Roitman *et al.*, 1998:227).

CAD is not necessarily inevitable just because of genetic predisposition and aging. There are several risk factors that cause variation amongst the prevalence of CAD (McArdle *et al.*, 1996:649; Roitman *et al.*, 1998:227).

Coronary risk factors

According to Plowman and Smith (1997:190) a risk factor can be defined as an aspect of a person's behaviour or lifestyle, environment, or and inherited characteristic that has been shown by epidemiological evidence to predispose an individual to the development of a specific disease.

During the past 30 years, various factors have been identified that are related to the susceptibility to CAD. These include personal characteristics and environmental factors.

Risk factors can be divided into modifiable and non-modifiable factors. Modifiable factors include hypertension, obesity, physical inactivity, cigarette smoking, nutrition, elevated cholesterol levels, diabetes mellitus, personality and behaviour patterns, high uric acid levels, pulmonary function abnormalities, electrocardiogram (ECG) abnormalities, tension or stress and poor education. Non-modifiable factors include age, gender, ethnic background, family history and male pattern baldness (McArdle *et al.*, 1996:650).

According to McArdle *et al.* (1996:650) it is not clear whether the modification of modifiable risk factors gives any protection from CAD. These authors do acknowledge, though, that the reduction or elimination of risk factors decreases the probability for the development of CAD.

Other authors (Tolfrey *et al.*, 2000) state that there is enough evidence from longitudinal paediatric population studies to make the direct link between risk factors in childhood and CAD later in life.

According to Thompson and Wilson (1992:4.1) the most important risk factors are those that are not, to our current knowledge, modifiable. Referring here to age, heredity,

gender and race. On the other hand, according to McArdle *et al.* (1996:651) the American Heart Association lists blood lipid abnormalities, obesity, cigarette smoking and hypertension as the "big four" CAD risk factors. Note that these are all modifiable.

1. Hypertension

Hypertension is often described as the "silent killer", because in most cases there are no symptoms (Ives, 2000). According to Kaplan (1983:61) 25% of all Americans suffer from hypertension. In South Africa, approximately 17% of the population has elevated blood pressure (Roussouw, 1990:50). Persistent hypertension affects 1-3% of children and 5-6% of adolescents (Strong, 1983:25). According to McArdle *et al.* (1996:275) each year an additional two million people are added to this figure.

Hypertension is defined as three consecutive blood pressure readings that are higher than normal or current use of anti-hypertensive medication (Kannel *et al.*, 1984; Kenney and Humphrey, 1995:206). Persons with blood pressure reading of 160/90mmHg or higher, have a 150-300% higher incidence for coronary artery disease and other related illnesses (Kannel *et al.*, 1984). Hypertension is predictive and causative of CAD, renal disease and stroke (Lauer and Clarke, 1980:281; Plowman and Smith, 1997:194). It is also associated with a 80% higher stroke mortality rate, a 50% higher CAD mortality rate, and a 320% higher rate of hypertension-related end-stage renal disease (ESRD) (Kountz, 2001).

1.1 Types of hypertension

There are two different types of hypertension, secondary hypertension and essential or primary hypertension. Secondary hypertension is when other diseases cause the raise in blood pressure, usually heart or kidney diseases. Essential or primary hypertension is when high blood pressure is caused by other unknown reasons (American Heart Association, 2000). Essential hypertension is the most common classification of hypertension. It is characterised by an increase in diastolic blood pressure and a related generalised arteriolar vasoconstriction that causes a rise in systolic blood pressure (Plowman and Smith, 1997:194; Roitman *et al.*, 1998:275).

Doctors and scientists used to think that all hypertension in children were secondary. Recently it is proved that children can get primary or essential hypertension (American Heart Association, 2000).

Raised systolic and diastolic blood pressures are both independent risk factors for CAD (Rossouw, 1990:51). Blood pressure levels in childhood are a very strong indication of what a person's adult blood pressure will be. That is why the knowledge of childhood blood pressure levels is of great importance (Gillman and Cook, 1995).

1.2 Classification of blood pressure

There are various stages of hypertension. The readings associated with the different stages are presented in table 2.2. The values shown here are applicable to all races, both sexes and throughout the entire adult life span (Plowman and Smith, 1997:193). Although mild hypertension is the most common form of hypertension, all stages of hypertension are associated with an increased risk for the development of CAD and renal disease. It is important to note that readings that are classified, as "high normal" is the first indication that lifestyle changes should be implemented (Kenney and Humphrey, 1995:206; Plowman and Smith, 1997:193).

Table 2.2 Classification of blood pressure for adults aged 18 years and older (Kenney and Humphrey, 1995:33; Plowman and Smith, 1997:194).

Category	**Systolic (mmHg)**	**Diastolic (mmHg)**
Normal	<130	<85
High normal	130-139	85-89
Hypertension		
Stage I (mild)	140-159	90-99
Stage II (moderate)	160-179	100-109
Stage III (severe)	180-209	110-119
Stage IV (very severe)	≥210	≥120

Numerous studies have shown the relationship between hypertension and serious medical conditions. According to Lauer and Clarke (1980:281) there have been no similar studies on children. According to these authors, the long-term significance of hypertension in children is uncertain.

The occurrence of hypertension in adults has been a well-known fact for years, but it was only recently that the focus has moved to children (Feinleib *et al.*, 1980:271). The classification of blood pressure for children and adolescents differ somewhat from the classification used for adults. The various stages of hypertension for children and adolescents are presented in table 2.3 and table 2.4, respectively.

Table 2.3 Classification of blood pressure for children aged 3-12 years (Plowman and Smith, 1997:194).

Category	**Systolic (mmHg)**	**Diastolic (mmHg)**
Hypertension		
Mild-moderate	≥130	≥86
Severe	≥144	≥96

Table 2.4 Classification of blood pressure for adolescents (Plowman and Smith, 1997:194).

Category	**Systolic (mmHg)**	**Diastolic (mmHg)**
Hypertension		
Mild-moderate	≥144	≥90
Severe	≥160	≥104

Note, that other authors categorised 10 to 15 year olds as children and older than 15 years as adolescents (Washington, 1999).

1.3 Hypertension as a coronary risk factor

Cardiac output and total peripheral resistance determine blood pressure. Hypertension causes an increased of workload on the heart, which results in increased left ventricular wall thickness and reduced early diastolic filling. There is a very strong correlation between left ventricle mass and CAD (Roitman *et al.*, 1998:276). According to Janz *et al.* (1995:818) left ventricle mass is one of the main predictors of blood pressure during childhood and adolescence. According to Paffenbarger and Lee (1996) hypertension is one of the main risk factors of cardiovascular disease.

Hypertension also determines the presence of coronary calcium, which is a predictor of sudden death. Another consequence of hypertension is stiffening of medium and small blood vessels (Roitman *et al.*, 1998:276).

1.4 Factors that influence blood pressure

1.4.1 Environmental factors

Numerous studies have found that environmental factors influence the blood pressure readings of adults. Only in recent years have studies begun to find a similar trend in children. Environmental factors have short- as well as long-term effects on a person's blood pressure. Factors that will have a short-term effect are, for instance, the effect of posture, exercise, ambient temperature, acute salt loads, bladder distension, and anxiety. Salt intake can also have long-term effects on blood pressure. Other environmental factors that can have long-term effects are social and psychological factors, changes in body weight and the use of various drugs (Feinleib *et al.*, 1980:271).

1.4.2 Genetic factors

Numerous studies have described the correlation between genetic influences and high blood pressure. Several twin studies have shown a correlation between the occurrence of hypertension in children and hypertension in their primary family members (Feinleib *et al.*, 1980:271-272).

It is a well-established fact that hypertension is more common amongst black people than white or coloured people. This is also true for children and adolescents (Rossouw, 1990:151; Thompson and Wilson, 1992:7.2; American Heart Association, 2000). According to the Roitman *et al.* (1998:275) the exact reason for differences in blood pressure of different races are not known.

1.4.3 Obesity

There is a direct relationship between obesity and hypertension (Bar-Or, 2000). In children and in adults, hypertension is more common among obese individuals (Dustan, 1980:305). Obesity doubles an individual's risk for future hypertension. It has the greatest impact before the age of 45 (Thompson and Wilson, 1992:7.3).

Weight gain in early adult life, are in most cases the beginning of the development of hypertension. No similar longitudinal studies have been done on children, so it is uncertain if childhood obesity increases the risk for later hypertension.

It is unsure by which mechanism obesity is related to hypertension. One of the theories is that the link is created by genetic factors.

It is important to note, that it is not just the overweight individual that is at risk, weight gain alone is also an important risk factor. The risk for developing hypertension is proportional to the degree of obesity (Dustan, 1980:305-306).

1.4.4 Dietary factors

For years, the effects of dietary salt on blood pressure have been debated. As early as in the beginning of the 20th century salt restriction was used to combat hypertension, mainly in France. Later in the 1920's the U.S. started to appreciate the benefits of restricted salt intake on hypertension. However, not all researchers were convinced. Some were concerned about the effects of prolonged salt restriction. There are many who still maintain that there is not enough scientific evidence to universally prescribe salt restriction (Anon, 1998).

It is a proven fact, that some people are genetically more sensitive to sodium intake than others (Page, 1980:291; Rossouw, 1990:64).

1.4.5 Age and gender

There is a sharp rise in blood pressure with age (Thompson and Wilson, 1992:7.1; Kenney and Humphrey, 1995:206; De Vries and Housh, 1996:123). Maturation also plays a role since there is a rise in blood pressure from childhood to adolescence. It is unclear if this rise in blood pressure is a natural occurrence of growth, or if it is the beginning of hypertension (Rossouw, 1990:54).

According to De Vries and Housh (1996:123) not all cultures show an increase of blood pressure with age.

Hypertension is more common in men than in women (Kenney and Humphrey, 1995:206). Blood pressure of women tends to be lower than that of men of the same age, before menopause. After menopause, the blood pressures of women tend to be slightly higher than that of their male counterparts.

2. Obesity

Obesity is a serious health problem. It is associated with an increased risk for mortality and morbidity, not just through CAD, but it also increases the risk for non-insulin dependent (Type II) diabetes, hypertension, and other illnesses. According to Paffenbarger and Lee (1996) obesity is one of the main causes of cardiovascular disease in adults. Because of this relationship, juvenile obesity has become a major public health challenge (Bar-Or, 2000). Although there has been a great increase in public awareness and concern for this problem, obesity remains a very common problem.

Obesity is described as an excess of adipose tissue-containing fat. The point at which excess fat can be classified as obesity is somewhat arbitrary (Roitman *et al.*, 1998:570). Kalk (2001) defined obesity "as a disease in which excessive body fat has accumulated to the extent that health may be adversely affected".

The most recognised fact of obesity is the frequent failure of treatment of this wide spread problem (Hirsch, 1975:15). Out of concern for adult obesity, several researchers have turned their attention to obesity in children (Stanley *et al.*, 1975:23). According to Heald (1975:81) obesity in children is totally different from obesity in adults. Juvenile obesity occurs in the developing phase of life and is characterised by hyperplasia of the adipose tissue.

The World Health Organisation (WHO) recently declared obesity as one of the major global health problems (Björntorp, 2001).

2.1 Classification

The American College of Sports Medicine (ACSM) defines overweight as a deviation in body weight from some standard or ideal weight related to height. According to this resource, "overweight" is when a person's weight is 20% above the ideal weight. It is very important to note however; that overweight does not necessarily reflects obesity.

An athlete may be, for instance, above the ideal weight, but be lean. It is thus not an easy task to classify body weight (Roitman *et al.*, 1998:570).

Numerous techniques have been developed to estimate body composition. It is important to note that none of the existing techniques actually measures the volume of fat, it is only an estimation of fat mass (Howley and Franks, 1997:168).

2.1.1 Body Mass Index

Body Mass Index (BMI) or Quetelet Index is the standard that is accepted by the World Health Organisation and is widely used for clinical assessment of appropriateness of body weight. It is described as a person's weight (kg) divided by height squared (m^2) (Howley and Franks, 1997:179; Roitman *et al.*, 1998:570). The BMI values that are used by the American College of Sport Medicine (ASCM) are presented in table 2.5.

Table 2.5 Classification of obesity according to BMI for males and females (Kenney and Humphrey, 1995:59).

BMI (kg/m^2)	**Category**
20-24.9	Desirable range
25-29.9	Grade 1 Obesity
30-40.1.1	Grade 2 Obesity
>40	Grade 3 Obesity (morbid obesity)

According to the National Centre for Health Statistics, health risks are increased at a BMI of 27, but a BMI of 25 indicates a potential need for intervention (McArdle *et al.*, 1996:54; Howley and Franks, 1997:179; Roitman *et al.*, 1998:570, Kalk, 2001; Van der Merwe, 2001). According to McArdle *et al.* (1996:542) the lowest health-risk category for BMI is between 20 and 25. Although a BMI of less than 20 does not increase the risk for CAD, it is associated with a greater risk for pulmonary and digestive diseases.

BMI does not differentiate between fat weight and lean weight (McArdle *et al.*, 1996:542; Howley and Franks, 1997:179). A high muscle mass, in relation to stature, because of genetic makeup or training, will result in a BMI reflecting "overweight". BMI should thus be viewed in relation to other variables (Howley and Franks, 1997:179).

2.1.2 Skinfolds

The use of skinfolds for the estimation of percentage body fat is one of the most frequent used methods. The appeal of this method is that it is quick, inexpensive and non-invasive (Howley and Franks, 1997:173).

Skinfold measures are used in prediction equations to estimate body density, which is then used to estimate body fatness (Janz *et al.*, 1993). Generalised equations have been developed for mature men and women (Kirkendall *et al.*, 1987:191).

The problem with children are, that before puberty children have more water and less bone mineral content, which results in over prediction of body fat (Janz *et al.*, 1993).

Therefore, formulating equations suitable for the estimation of percentage body fat in children became a priority. Lohman (1992:66) started by using 10 skinfolds. This was later reduced to two skinfolds. Today, the use of two skinfolds for the estimation of percentage body fat in children is widely accepted. Părizjková (1961) reported correlations of 0.81 to 0.95 between hydrostatic weighing and tricep and subscapular skinfolds in nine to 16-year-old children.

2.1.3 Waist-hip ratio

It is not just the percentage of fat that is of importance, but also the distribution. The localisation of excess fat indicates different risks (McArdle *et al.*, 1996:609; Björntorp, 2001). Waist-hip ratio is an excellent predictor of CAD mortality and morbidity, stroke and diabetes mellitus (Norton and Olds, 1996:382).

There are two types of regional fat distribution, central and peripheral obesity. Central obesity is characterised by fat deposition mainly in the abdominal region, whereas peripheral obesity is characterised by fat deposition mainly in the femoral and gluteal regions. Central obesity is associated with an increased risk for hyperinsulinemia, insulin resistance, Type II diabetes, endometrial cancer, hypercholesterolemia, hypertension and atherosclerosis. Central obesity is thus associated with an increased risk for CAD. Peripheral obesity is associated with less serious conditions that are caused by mechanical problems associated with the increased weight. The importance of distinguishing between these two types of obesity is clear, which is where the waist-hip ratio becomes important.

Waist-hip ratio is the ratio between the smallest circumference of the waist and the biggest circumference of the hips. There are ethnic variations in these measurements, but the limits most often used are >0.95 for men and >0.8 for women (McArdle *et al.,* 1996:609; Björntorp, 2001).

2.2 Prevalence

The number of overweight people has increased at an alarming rate during the last 20 years. In the United States of America (U.S.A.), more than half of the population over the age of 20 is overweight. The rest of the world is following the same trend. In Europe it is estimated that 20.5% of men and 26.8% of women are overweight (Björntorp, 2001; Van der Merwe, 2001). According to Björntorp (2001) the prevalence of obesity amongst eastern European women is as high as 60-70%. Howley and Franks (1997:184) stated that approximately 58 million American adults are obese. The American College of Sports Medicine (Roitman *et al.*, 1998:570) estimated the number of obese adults in the U.S.A. as 34 million.

The most recent data available for South Africa was data collected in a national demographic and health survey conducted in 1998. Of all the men between the ages of 15 and 24, 11.1% have a BMI of ≥ 25 and 2.7% have a BMI of ≥ 30. For men between the ages of 25 and 64 the figures are more alarming. Forty four percent of these men have a BMI ≥ 25 and 15% have a BMI ≥ 30 (Kalk, 2001). Obesity is prevalent amongst

19.8% of white men, 19% of black men, 8% of coloured men and 4% of Indian men. The prevalence of overweight is the lowest amongst black men (20.3%) and the highest amongst white men (54.1%) (Kalk, 2001; Van der Merwe, 2001).

For South African women it is a totally different picture. Twenty nine percent of women between the ages of 15 and 24 are overweight, 9.4% of them are obese. For women under the age of 65, an alarming 70% are overweight and 44% are obese (Kalk, 2001). Obesity has the highest prevalence amongst black women (55%), followed by coloured women (43%), then Indian women (36%) and lastly white women (32%) (Van der Merwe, 2001).

According to McArdle *et al.* (1996:603) an alarming 45% of all American children are obese. The WHO estimated that approximately 10% of schoolchildren in Europe are obese (Gleick, 1999). Childhood obesity is a common phenomenon in South Africa (Kalk, 2001). According to a health and fitness survey 35% of all South African girls will be overweight by the time they reach 18 (Sunday Times, 2002).

2.3 Aetiology

The biggest problem with obese children is the potential of them becoming obese adults (Bar-Or *et al.*, 1998; Rolfes *et al.*, 1998:235, Kalk, 2001). According to Bar-Or (2000) 50% of all obese children older than six years are more than likely going to be obese adults. Rolfes *et al.* (1998:235) divide problems that stem from obesity into three categories: growth, physical health, and psychological development. Concerning growth, these authors estimate that obese children develop characteristic physical traits. Typically these children begin puberty earlier than their peers. The relationship between obesity and maturation is not clearly understood. Girls that mature earlier tend to be shorter and fatter than what their peers are.

The physical health category is what this study is more concerned with. Like in obese adults, the blood lipid profiles of obese children are also of some concern. Lipograms often display high levels of triglycerides, low-density lipoprotein (LDL) and total

cholesterol. As stated above, these children also have a tendency to have hypertension.

Kalk (2001) ascribes the prevalence of obesity amongst pre-adolescents to poor social functioning and impaired academic performance. This author associates obesity amongst adolescents with low self-esteem, social and economic problems, physical disabilities and type II diabetes.

Obesity is not caused by a single factor, which makes defining the causes a difficult task. A positive energy balance (higher energy intake than expenditure) leads to obesity. A combination of factors usually contributes to the development of obesity. (Howley and Franks, 1997:185; Björntorp, 2001).

2.3.1 Heredity

The impact of genetics on the development of obesity is two-fold. There are factors that are truly genetic and then there are social-cultural factors that are passed on through the generations. Bouchard *et al.* (1988) estimated that percentage body fat is 25% accounted for by inheritance.

There is a significant increase in the risk of becoming an obese adult for children with one or two obese parents (Kalk, 2001). An obese adolescent has a 70% chance of becoming an obese adult. If one parent is obese, this risk increases to 80% (Mayer, 1975:76; Bar-Or, 2000). Mayer (1975:76) found no correlation between the weight of children who were adopted at birth and the weight of their adoptive parents.

Animal studies have found that obesity in animals can be caused by mutations in regulatory mechanisms, especially the leptin system. Leptin is a protein, mostly secreted by adipose tissue, which regulates energy intake through a receptor in the hypothalamus. Mutations of the leptin- and leptin receptor gene have been found in obese animals. Leptin gene abnormalities in humans are very rare. Although research is inconclusive, it seems that some humans are resistant to the actions of leptin, because of obesity in spite of high leptin levels (Björntorp, 2001).

2.3.2 Sedentary lifestyle

A sedentary lifestyle is probably the most recognised lifestyle risk factor, which contributes to excessive weight gain (Kalk, 2001). It is generally accepted that obesity and inactivity goes hand in hand (Parr, 1998).

The relationship between obesity and inactivity is complex, especially in young children. It has, however, been proved that increased activity together with restriction of calorie intake is effective for weight management in children (Ganley and Sherman, 2000). Overweight children are least likely to engage in physical activity, which contributes further to the relationship between inactivity and obesity (Carlson, 2000).

A recent study done by Zakas and co-workers (2001) showed a significant decrease ($p<0.001$) in tricep, subscapular and abdominal skinfold thickness of 10, 13 and 16-year old boys after a twelve-week training period. The exercise programme consisted of 50 minutes cycling, on a cycle ergometer, three times a week.

In a study done by Mayer (1975:77) she found that most of the weight children gain, is gained in winter. The conclusion was made that activity was the determining factor. It was found that obese girls are 33% less active than non-obese girls. This study also looked at the time children spent in motion while exercising. On average, non-obese children are in motion about 90% of the time when exercising, while obese children are only in motion 50% of the time.

A recent study in the United States found that television watching is associated with obesity amongst girls. The prevalence of obesity was the highest amongst children who watched television for more than an hour a day (Anon, 2001a).

2.3.3 Food intake

According to Mayer (1975:73) 500g excess adipose tissue is equivalent to 3500 calories. This means that a child who eats 100 calories a day more than what is expended, there would be a weight gain of five kilograms in a year. When looking at the

food intake of the obese child, this is very important. The difference in food intake between the non-obese child and the obese child would thus be small, but will still cause excess weight gain.

It was found that obese children do not always eat more than non-obese children do. In some instances they tend to eat less, but their energy expenditure was far less.

Animal studies done by Mayer (1975:77) found that animals do not become fat because they eat too much carbohydrate. It was true however, for animals on low-carbohydrate diets and high-fat diets.

2.3.4 Parental neglect

A risk factor that is of great concern in South Africa is parental neglect. It is a known fact that parental neglect greatly increases a child's risk of obesity in early adulthood (Kalk, 2001).

2.3.5 Socio-economic status

Another factor contributing to obesity is socio-economic status. It is a known fact that obesity is more common in lower socio-economic groups (Crowther and Van der Merwe, 2001).

According to Mayer (1975:76) the relationship between socio-economic status and obesity is much more complex. In her research Mayer (1975:76) found that there is an inverse relationship between obesity and socio-economic status for women. For men however, the prevalence of obesity was very low on the top and the bottom of the socio-economic scale, with the highest prevalence of obesity in the middle. This is probably because men at the bottom of the socio-economic scale usually perform heavy work and the men at the top can afford exercise equipment and gymnasium memberships.

Mayer (1975:76) found no correlation between obesity and socio-economic status in children. However, a correlation was found amongst girls in their puberty.

2.3.6 Stunting

Childhood obesity amongst black South African children may be associated with "stunting". "Stunting" is when a child is too short for his/her age but also too fat for his/her age. This can be improved by zinc supplementation (Kalk, 2001). According to a survey done in 1994 the prevalence of childhood stunting in South Africa is 22.9% (Anon, 1999).

2.3.7 Obese hypoglycaemic syndrome

There are a number of metabolic diseases that can cause obesity. One of these is the obese hypoglycaemic syndrome that is characterised, in addition to extreme obesity, by hypercholesterolemia and diabetes (Mayer, 1975:75).

2.3.8 Body type

According to Mayer (1975:75) certain body types are associated with a high prevalence of obesity and other body types never become obese. According to this author the ectomorph body type is not associated with obesity. She goes as far as saying that these people do not even get fat. One of the reasons could be that ectomorphs do not have enough adipose tissue to accumulate fat.

2.4 Economic costs

The economic burden of obesity is two-fold; there are direct and indirect costs involved. Direct costs are costs linked directly to the treatment of obesity and its co-morbidities. Whereas indirect costs are accounted for by financial loss due to absenteeism, sick leave and early retirement (Björntorp, 2001; Kalk, 2001; Van der Merwe, 2001).

According to Howley and Franks (1997:184) approximately $30 billion is spent annually in the U.S.A. on weight loss efforts. McArdle *et al.* (1996:603) estimated this annual

figure to be as high as $35 billion. According to Gleick (1999) the latest estimates show that annual healthcare cost for obese adults is as high as $238 billion.

Van der Merwe (2001) stated that 7-8% of westernised countries' total health expenditure is accounted for by direct costs of obesity. As BMI increases, these costs rise as well. Relative to a BMI of 20-24.9, with a BMI of 30-34.9 and a BMI of 35 or greater the mean annual costs are 25% and 44% greater respectively. The indirect costs of obesity, such as lost of productivity, for the U.S.A. are estimated at $70 billion annually.

The economic burden of obesity is higher than the costs involved in all cancer treatments or AIDS (Björntorp, 2001).

At the 2001 congress of the South African Society for the Study of Obesity it was stated that obesity epidemic has the potential to bankrupt health care systems (Anon, 2001b).

2.5 Role of schools

Ninety five percent of children ages five to 17 are enrolled in school. Most children eat one or two meals at the school. The importance of school-based intervention programs is thus clear (Bar-Or *et al.*, 1998).

3. Physical inactivity

According to Katzmarzyk and associates (2001), there is extensive evidence that a sedentary lifestyle, poor aerobic fitness and associated obesity are primary risk factors for CAD. It is an established fact that habitual physical activity reduces the risk for the development of CAD (Young and Steinhardt, 1993). High physical fitness levels are protective of all-cause mortality, even in overweight individuals. Low physical fitness levels, and not an elevated BMI (>26 kg/m^2) as such, are associated with an increased risk for CAD mortality (Katzmarzyk *et al.*, 2001). Besides being an independent risk factor for CAD, physical inactivity is also a risk factor for hypertension, hypercholesterolemia and obesity (Washington, 1999). According to Boreham *et al.*

(2001) there is an inverse association between aerobic fitness and CAD mortality and risk factor status. In adults, these relationships are independent of other risk factors, including obesity. In children, however, this relationship appears to differ. High aerobic fitness levels still have a positive effect on CAD risk, but this relationship may mediate from body fatness and not aerobic fitness as such.

There are a number of mechanisms by which physical activity can reduce the risk of developing CAD. Physical activity causes changes in the cardiovascular system and its neural control. Through the increasing of parasympathetic tone and decreasing of sympathetic response it causes a lower resting and exercising heart rate. These changes enhance the electrical stability of the myocardial cells, which reduces the risk of fatal coronary spasms and conduction system defects. The other mechanisms through which physical activity reduces the risk of CAD are linked to other risk factors (Plowman and Smith, 1997:195).

The International Consensus Conference on Physical Activity has defined physical activity as "any bodily movement produced by skeletal muscles that results in energy expenditure" (Myers *et al.*, 1996). It is important to differentiate between "physical activity" and "physical fitness". Most studies look at the relationship between CAD and physical activity, but some studies found a stronger relationship between CAD and physical fitness than physical activity (Young and Steinhardt, 1993). Young and Steinhardt (1993) found in their research that low physical activity and low physical fitness are associated with an increased risk for CAD. Anderson and Haraldsdottir (1995) stated that most studies found a closer relationship between physical fitness and CAD risk factors than physical activity and CAD risk factors. The study done by Anderson and Haraldsdottir (1995) found a weak but significant relationship between direct measurements of $\dot{V}O_2max$ and physical activity. They found however, no relationship between physical activity and CAD risk factors, but there was a clear relationship between $\dot{V}O_2max$ and CAD risk factors. Results regarding physical activity versus physical fitness and the influence on CAD risk factors are thus somewhat contradictory. The different ways of assessing physical activity and physical fitness respectively should be evaluated to give more accurate results for future research. The National Heart, Lung, and Blood Institute (1998) recommend better methods of physical

activity assessment, better definitions of physical activity and physical fitness, standardisation of methods for data collection.

The health benefits of physical activity are well known and accepted. Most research however, has been based on adults. More research is needed on the current levels, types, intensity and patterns of activity in children (Myers *et al.*, 1996).

According to Ganley and Sherman (2000) two common misconceptions amongst adults contribute to the lack of physical activity amongst children. Firstly, most people assume that children are naturally and spontaneously active and secondly, it is assumed that health risks associated with the lack of physical activity are only applicable to adults. There is enough evidence that contradicts the above assumptions.

3.1 Prevalence

More than 50% of the children in the United States are not getting enough exercise for cardiovascular and health benefits. There is also enough evidence that shows that activity levels of children decline with age, especially when entering puberty. One fourth of all children between the ages of 12 and 21 in the United States engages in no physical activity (Ganley and Sherman, 2000).

Physical inactivity is more prevalent amongst girls than boys, during and after puberty (Ganley and Sherman, 2000). According to a study done by Myers and co-workers (1996) regarding children between the ages of nine and 15, boys are on the average more active than girls. This study also found differences in activity levels between different ethnic groups, with white children having higher activity levels than black children. It should be noted however, that the physical demands of daily living of the black children were higher than that of the white children. This probably explains why they did not engage in so much organised physical activities.

In a study done by Fardy *et al.* (2000) they found that 64% of the teenage girls and 45% of the teenage boys tested, felt that they are not getting enough exercise, 26% of the girls and 15% of the boys reported less than 30% of activity in a week.

3.2 Aetiology

Physical inactivity is one of the factors contributing to obesity, the reason why these two factors are so closely linked.

It has been shown that physical activity patterns track from adolescence to adulthood, which means that active adolescents are likely to become active adults, and visa versa (Myers *et al.*, 1996).

3.2.1 Television watching

Television watching is the most prevalent sedentary behaviour amongst children in the United States (Bar-Or *et al.*, 1998). Surveys done in the United States pointed out that on the average, children between the ages of 10 and 17 spend about three hours a day in front of the television (Myers *et al.*, 1996). The typical 21 hours of television watching should ideally be reduced to seven hours. This will cut a child's risk of becoming obese by one third. For every five hours a week added to time spend in front of the television a child's risk of becoming obese is increased by 10% (Parr, 1998).

Television watching is associated with reduced energy expenditure, increased consumption of food while watching television, and an increase of consumption of food advertised on the television (Bar-Or *et al.*, 1998).

With the increase in technology there is an increase in accessibility of computer games for children. This causes an increase in sedentary activity (Bar-Or *et al.*, 1998).

3.2.2 Physical education

Conclusive evidence for the long-term benefits of physical activity was only provided late in the 20th century. At the same time there was a newly found interest in the virtues and essentiality of physical education and the role it plays in laying the foundation for physical activity (Hardman and Marshall, 2001:15).

"I don't like it" and "I'm not motivated" are two of the most common reasons teenage girls give for not being active. This emphasises the need for good school-based programmes that integrate vigorous physical activity. Unfortunately physical education in schools has declined in the past decade with 50% (Fardy *et al.*, 2000). Many schools do not consider physical education a priority. Thirty seven percent of all countries view physical education as a non-essential part of the school curriculum (Hardman and Marshall, 2001:15-16). Budget restraints in schools are forcing schools to eliminate physical education classes (Bar-Or *et al.*, 1998).

Only about one third of primary schools in the United States offer physical education classes. Enrolment in physical education classes among high school students in the United States has declined from 42% in 1991 to 25% in 1995 (Parr, 1998; Ganley and Sherman, 2000).

3.2.2.1 Physical education in South Africa

The first democratic election in South Africa was held in 1994. This marked the start of numerous changes and reconstruction in all systems, including education.

In the 1980's many of the historically disadvantaged schools started to phase out physical education and structured sport activities due to transport and financial difficulties. As a result, many children in Western Cape schools have never experienced physical education or structured sport activities (Centre for Education Policy Development, Evaluation and Management & Education Policy Unit, 1999:43). Before 1994, 85% of all primary schools in South Africa had no physical education (Keim and Zinn, 1998). The poor status of physical education in South Africa can largely be ascribed to disparities of the past and transformation processes in the educational system. Transformation processes included new teacher-learner ratios, as the class sizes increased. As a result, physical education was limited to grade eight and nine level or, in many cases, eliminated all together (Centre for Education Policy Development, Evaluation and Management & Education Policy Unit, 1999:48).

In 1997 Curriculum 2005, a system based on the principles of Outcome Based Education, was introduced in South African schools. Physical education is incorporated under "Life Orientation" together with four other focus areas (Centre for Education Policy Development, Evaluation and Management & Education Policy Unit, 1999:13 & 44). As a result, physical education specialists are no longer appointed at schools. Teaching of physical education is now left to general teachers who might have no skill or understanding of it (Hardman and Marshall, 2001:27). Adding to the poor status of physical education, is the fact that there is no clear description or structured programme for it incorporated in Curriculum 2005.

Physical education is only one subdivision of "Life Orientation", which causes a significant decrease in contact time. In most cases there is no physical education for pupils in grade 12. For grade eight to grade 11 there are, in most cases, only 30 minutes a week allocated for physical education (Katzenellenbogen, 1995). Although physical education is a very low priority, it is still compulsory at all schools up to grade nine. There are however, no monitoring strategies in place to ensure that schools follow these regulations (Centre for Education Policy Development, Evaluation and Management & Education Policy Unit, 1999:47).

3.2.3 Genetics

Looking at fitness scores, thus physical fitness and not physical activity, genetics have a definite impact (Eaton *et al.*, 1995; Plowman and Smith, 1997:200). Unfortunately it is true that some individuals will score high on fitness tests without doing any physical activity. The opposite is also a reality; some individuals will never score very high on fitness tests, despite being active (Plowman and Smith, 1997:200).

3.2.4 Emphasis on academic achievements

Parents and the education system are placing more and more pressure on children to excel in academics. This can cause some children to be less concerned about positive

exercise behaviour and concentrate more on scholastic achievements (Schmidt *et al.*, 1998).

3.2.5 Environmental factors

Various environmental factors, such as heat, humidity or rainy conditions can force children to stay indoors. A lack of facilities to practise sport or any physical activities indoors can contribute to sedentary behaviour in children (Schmidt *et al.*, 1998). Liability and insurance issues in schools are causing schools to limit after-hour use of facilities by the community (Bar-Or *et al.*, 1998).

Safety is also an issue, especially in urban settings. In the day and age we live in it is not possible in all settings for children to walk around and play outside by themselves. Fenced-in backyards and traffic are also contributing to children staying indoors. For safety and comfort, children are also being driven to school. Smaller families are also contributing to more sedentary children (Bar-Or *et al.*, 1998).

4. Cigarette smoking

Approximately 20% of deaths from CAD can be directly linked to cigarette smoking. Secondary smoking also increases the risk for the development of CAD (Plowman and Smith, 1997:193).

There are a number of ways that smoking can be detrimental to a person's health and contribute to CAD risk. Nicotine stimulates the sympathetic nervous system. This causes an acute rise in heart rate and blood pressure that increases the workload on the heart. Carbon monoxide binds with haemoglobin, which causes oxygen transport to be reduced. There are a couple of ways that smoking speeds up the atherosclerotic process. Firstly, smoking injures the arterial wall lining, which usually marks the beginning of atherosclerosis. It also increases the levels of circulating total cholesterol and reduces the amount of high-density lipoproteins. Smoking also causes blood platelets to stick to each other. Further more, it accelerates the rate of internal blood clotting and these clots that form are very tough and difficult to dissolve. Smoking

reduces prostacyclin, which is responsible for the dilation of blood vessels. This causes the capillaries and small arteries to constrict, thus increasing the likelihood of a blood clot to block one the arteries. Smoking can also cause life-threatening arrhythmias. It is thus clear that smoking is an independent risk factor, but it also contributes to other CAD risk factors (Plowman and Smith, 1997:193).

4.1 Prevalence

The drastic decline in tobacco use in European, Scandinavian and North American countries has led to aggressive marketing by tobacco companies in developing countries, which explains the increase in tobacco sales in the developing world. Children in developing countries will thus make up the majority of future smokers, but to date, little attention is paid to these children or smoking in general in developing countries (De Wet *et al.*, 2000).

Cigarette smoking amongst teenagers is rising sharply (Killen *et al.*, 1988). In the United States it is estimated that 2.2 million of adolescents between the age of 12 and 17 smoke (Plowman and Smith, 1997). According to Washington (1999) 8.5 million adolescents between the ages of 12 and 17 have tried cigarette smoking. Everyday another 3000 youngsters are added to this alarming number. Seventy five percent of adult smokers started smoking before the age 18, 90% of them started smoking before the age of 21 (Plowman and Smith, 1997:198; Washington, 1999).

The mean age for first tobacco use is 10.7 years for boys and 11.4 years for girls (Washington 1999). According to studies in China and India, children from developing countries are starting smoking at an increasingly younger age (De Wet *et al.*, 2000).

It is estimated that one in three South African adults smoke. Tobacco use has increased by 3% in South Africa since 1992. In a study done by De Wet and associates (2000) on five year olds in South Africa, they found that seven percent of the subjects have tried smoking at five years of age. Nineteen percent of these children thought that they would smoke when they grow up.

4.2 Aetiology

Adolescents take up smoking for numerous reasons. Peer influence seems to play the most important role in the initiation of smoking. It is an activity that can make teenagers feel mature and more accepted by peers. Among teenage girls the most common reason for smoking is to lose weight (Washington, 1999).

4.3 Secondary smoke

Secondary smoke is considered a coronary risk factor (Plowman and Smith, 1997:193; Steyn *et al.*, 2000). Exposure to secondary smoke on a daily basis not only increases a child's coronary risk, but also contributes to the development of respiratory diseases. It has also been shown that children that come from a household were the parents or caretakers smoke, are more likely to take up smoking at some stage in their life (Steyn *et al.*, 2000).

In the study done by Steyn and associates (2000), they found that coloured children in South Africa have the highest rate of exposure to secondary smoke. In their study the white children were least exposed to secondary smoke.

5. Nutrition

Diet is associated with eight of the ten leading causes of death in the United States (National Heart, Lung and Blood Institute, 1998). The American College of Sports Medicine (Roitman *et al.*, 1998:5) classifies diet as one of the most important environmental causes of CAD.

The National Heart, Lung and Blood Institute (1998) classify nutrition as an independent risk factor for CAD. According to their research increased levels of homocysteine can be directly linked to the development of CAD. The ingestion of antioxidants, folate, vitamins B6 and B12, and soluble fibre can lower levels of homocysteine, and thus lower the risk for CAD (National Heart, Lung and Blood Institute, 1998; Roitman *et al.*, 1998:5).

Diet further increases the risk for the development of CAD by contributing to hypertension, obesity and diabetes mellitus. High intake of saturated fat is also associated with an increased risk for thrombogenicity (Roitman *et al.*, 1998:5).

5.1 Elevated cholesterol levels

Elevated cholesterol levels seldom occur in isolation, except when heredity is the cause of hypercholesterolemia. High saturated fat and high cholesterol diets increase total and low-density lipoproteins that increase the risk for CAD (National Heart, Lung and Blood Institute, 1998; Roitman *et al.*, 1998:5).

Elevated cholesterol levels in childhood play a big role in the development of adult atherosclerosis. According to Killen *et al.* (1988) 40% of children's energy intake is from fat. Fifteen to eighteen percent of fat intake consists out of saturated fats and the estimated average cholesterol intake of 300 $mg.d^{-1}$ is well in excess.

6. Diabetes mellitus

Diabetes mellitus is a metabolic disorder characterised by glucose intolerance. Simply put, glucose intolerance is the body's inability to effectively use carbohydrates. There are two different types of diabetes. Type I or insulin dependent diabetes is common in young individuals. As the name indicates, these individuals are dependent on an external source of insulin. It occurs when the insulin-producing Beta cells of the pancreas fail. Type II or non-insulin dependent diabetes is common amongst overweight or obese adults over 40 years of age. In these cases there are usually sufficient or excess levels of insulin produced. The problem is that the sensitivity to the circulating insulin is discontinued, especially in the skeletal muscles and liver.

Among the many pathological complications, diabetes also causes an acceleration of atherosclerosis (Plowman and Smith, 1997:196). The risk for the development of CAD is increased two- to fourfold by diabetes mellitus. CAD and atherosclerosis are the cause of 80% and 75% of diabetic mortality. In the United States, 25% of persons who suffer myocardial infarctions are diabetics (Roitman *et al.*, 1998:8).

Women with diabetes mellitus are at greater risk for the development of CAD (Roitman *et al.*, 1998:343).

6.1 Type II diabetes

According to Rapaport (2001) an elevation of plasma free fatty acids can be the cause of Type II diabetes. Free fatty acids inhibit the actions of insulin and influence the transport of glucose to skeletal muscles.

Risk factors for Type II diabetes includes obesity, puberty at 13.5 years, female gender and a family history of Type II diabetes (Rapaport, 2001).

7. Family history

Family history is classified as a CAD risk factor if there is any primary family member with premature (younger than 55 years of age for males and 65 years for females) CAD or death from CAD, especially heart attack or stroke. If there is a family history of diabetes mellitus, hypertension, and/or hyperlipidemia it also increases the risk for CAD (Plowman and Smith, 1997:190).

According to the National Heart, Lung, and Blood Institute (1998) there are enough studies on heredity and CAD risk factors to conclude that there is a significant heredity component to CAD risk.

8. Gender

According to the Roitman *et al.* (1998:228) men are at greater risk for developing CAD. The reason for this could be that in earlier years, women did not work and did not experience the stress that goes with that. Smoking and alcohol use were also more common amongst men than women. With that changing, women are also becoming at greater risk.

According to Plowman and Smith (1997:190) being female is not an automatic protection against CAD, although many women are led to believe that. The number of deaths from CAD for 1984 to 1990 was higher for women than for men. This could, however, just be a reflection of the higher population females than males. Research has shown that women who suffer from heart attacks are more likely to die within a few days to a year, or suffer a second heart attack.

A study done by Fardy *et al*. (2000) found that teenage girls have a higher prevalence of CAD risk factors than teenage boys. This is due to girls on average had higher percentage body fat, greater total cholesterol, lower physical activity and poorer cardiovascular fitness. This study found that the girls were more informed and knowledgeable about better health behaviours and risk reduction, which indicates that more health knowledge does not necessarily translate to better health behaviour. CAD risk reduction was in the past neglected as part of women's health. This is because CAD is seen as a "male disease". The above findings indicate, however, that it should be an integral part of not just men's, but women's health as well.

CHAPTER III METHODS

Study population

Boys and girls in grades five to grade seven were selected as the study population. This age group was selected based on the fact that early recognition of risk factors would leave time for intervention strategies to be implemented that could aid the prevention of CAD later in life.

Sample

A list of all primary schools in the Western Cape was obtained from the Western Cape Schools Board. The area defined as the Western Cape is illustrated in Figure 3.1. A letter explaining the importance of the study (Appendix A) was sent to 15 schools that were randomly selected from the list. Out of the 15 schools, only four schools replied.

It was decided that grade five to seven would cover the intended age groups. Each school decided how much time they would allocate for the testing of each grade individually. A second letter (Appendix B) was sent to these four schools informing them about testing procedures and testing dates. Questionnaires and letters of informed consent (Appendix C) were sent to each of the schools, which were completed by the parents of the children in the mentioned grades. Only children whose parents completed these forms were tested.

Based on the time allocated to the study by each school and consent by the parents, the sample consisted out of 288 children, who were made up of 78 grade five, 104 grade six, and 106 grade seven children. The gender spilt of the sample was 154 boys and 134 girls.

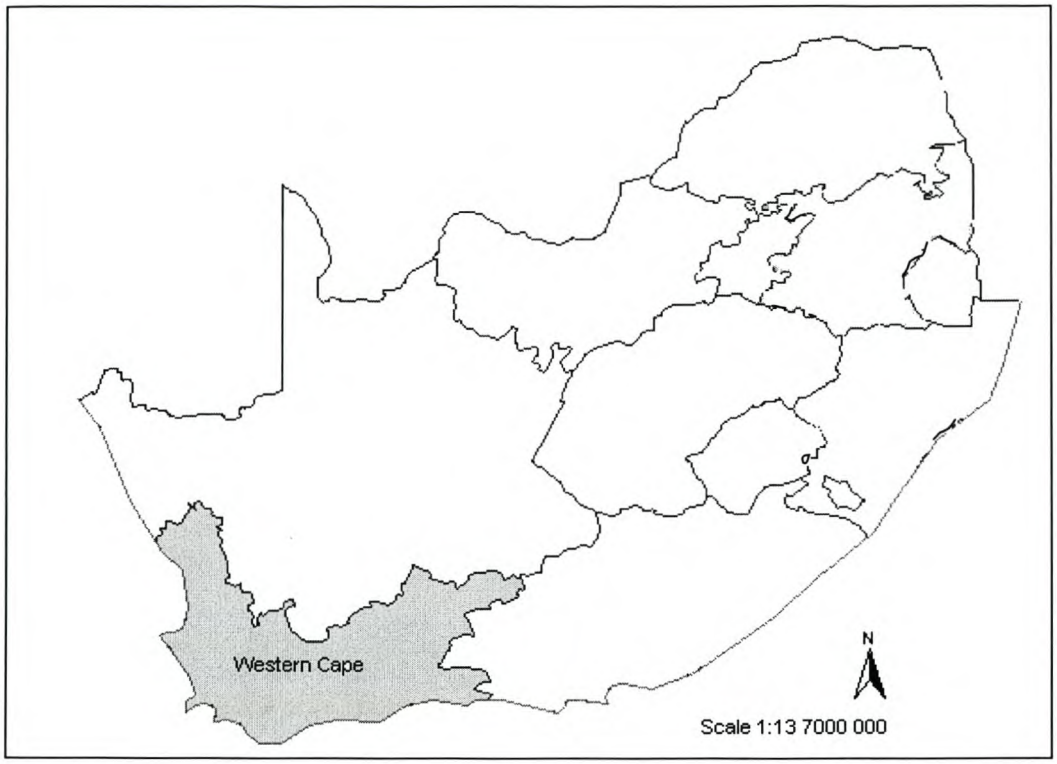

Figure 3.1 Regional map of South Africa indicating the Western Cape Province.

Survey procedure

The survey was conducted in August and September 2001. The team collecting the data consisted of two Biokinetic interns and eight fourth-year Biokinetic students. The assistants in this study were experienced in clinical testing and familiar with the protocols and equipment being used. The same assistants were used for all the measurements.

Once the children were called from their classrooms, each child received a data form (Appendix D) with a code number. To speed up this process, all forms were numbered in advance. Each child filled out section A and C of the forms with the help of one of the assistants. Personal details were filled in first (Section A). Without the children knowing, the assistants documented race. Section C was a questionnaire that assessed activity levels.

After personal details and activity levels were documented, the girls and boys were separated and divided into smaller groups. The children were asked, in advance, to wear light clothing (shorts and short-sleeved shirts). Each girl was given a short, cropped top to wear to ease skinfold measurement. Each group went to a different station, where depending on the station, different data was collected. After completion of one station the group progressed to the next. The step test was conducted right at the end, after all stations were completed.

To minimise anxiety, girls and boys were always kept apart, either in different classrooms or by dividing the room with a curtain. Anthropometrical testing procedures as described by Norton and Olds (1996:35-58) were followed.

1. Stature

Standing height, without shoes, was measured to the nearest millimetre with the use of a stadiometer.

The subjects stood erect with heels together and arms hanging by the sides. Using both hands, the subject's head was cupped along the mastoid processes to correctly position it in the Frankfort Plane. The Frankfort plane is achieved when the lower edge of the eye socket is in the same horizontal plane as the notch superior to the tragus of the ear. The subjects were instructed to look straight ahead and to take a deep breath. The measurement was taken as the maximum distance from the measuring platform to the vertex of the head, at the end of the deep inward breath. The vertex of the head is defined as the highest point on the skull when the head is held in the Frankfort Plane (Norton and Olds, 1996:37).

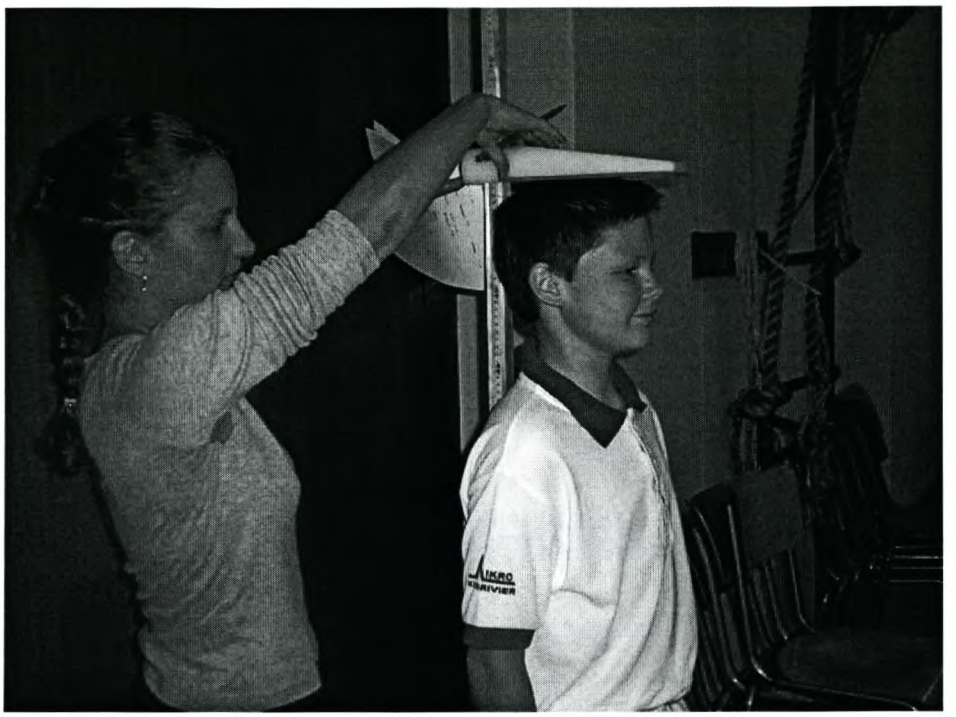

Figure 3.2 Measurement of stature with the use of a stadiometer.

2. Body weight

Body weight was determined with the use of a UWE BW-250 electronic scale to the nearest 0.1kg. The scale automatically calibrated itself after each measurement. Subjects were weighed without shoes, wearing light clothing.

3. Body Mass Index (BMI)

By using stature and body weight measurements, BMI was calculated using the standard formula (Appendix G).

4. Waist-hip ratio

Waist-hip ratio was determined by calculating the ratio between the waist and hip circumference.

4.1 Waist circumference

Waist circumference was measured with subjects standing erect with arms relaxed at the sides, at the end of normal expiration. The measurement was taken at the narrowest point between the last rib and the iliac crest. Where it was difficult to locate the narrowest point, the measurement was taken at the mid-point between the last rib and the iliac crest.

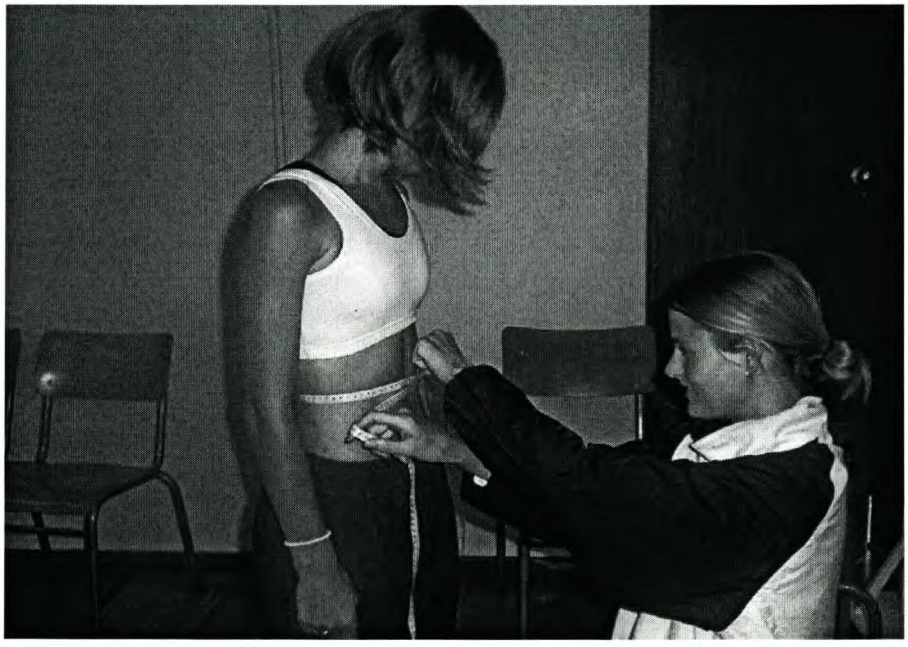

Figure 3.3 Measurement of waist circumference.

4.2 Hip circumference

Hip circumference was taken while subjects were standing erect with feet together and gluteal muscles relaxed. The measurement was taken at the greatest posterior protuberance of the buttocks. When taking this measurement, the measurer was standing at the subject's side to ensure the tape is horizontal.

5. Skinfolds

Skinfolds were measured with a Harpender skinfold caliper. Two skinfolds were measured, tricep and subscapular, as described by Pařízková (1961), Lohman (1987) and Slaughter *et al.* (1988). Fat percentages were calculated using Slaughter's formula (Appendix G).

Before taking the skinfold measurement both sites were marked with an eye pencil. Both skinfolds were taken on the right side of the body. Measurements were recorded two seconds after the full pressure of the calipers were applied. A minimum of two measurements was taken at each site. The average of the two measurements was used, if the two measurements were within 0.4mm of each other. If there was a difference greater than 0.4mm, measurements were retaken after a few minutes.

The procedure was fully explained to the children and they were informed that more than one measurement was going to be taken. In cases where a child was very anxious a measurement was first taken on the left side of the body. This ensured that the child did not jerk when taking the real measurement.

5.1 Tricep skinfold

First, the point at the superior and lateral border of the acromion process (acromiale) was identified and marked, as well as the point at the proximal and lateral border of the head of the radius (radiale). Next the mid-acromiale-radiale point was identified and marked with the arm relaxed and extended by the side. The mid-acromial-radiale point is defined as the mid-point between the acromiale and radiale. A vertical fold was taken

on the most posterior surface of the tricep muscle on the mid-acromiale-radiale line. Subjects were instructed to keep their arm relaxed, with the shoulder slightly externally rotated and the elbow extended by the side of their body.

Figure 3.4 Measurement of tricep skinfolds with a Harpender skinfold caliper.

5.2 Subscapular skinfold

First, the most inferior tip of the inferior angle of the scapula was identified and marked, with the subject standing erect with arms at the side. The measurement was taken two centimetres along the line running laterally and obliquely downwards from the marked point at an approximately 45° angle.

6. Blood pressure

Blood pressure was measured using a sphygmomanometer. Depending on the size of the child's arm, a standard paediatric cuff (7 x 21cm) or an adult cuff (12 x 23.5cm) was used.

Subjects had to sit down and relax for at least five minutes before blood pressure was measured. It was difficult to get the subjects to relax and sit still for the five minutes when other children were around, especially the boys. To overcome this problem blood pressure was measured in a separate room.

The systolic reading was taken at the point where the first Korotkoff sound was heard and the diastolic reading was recorded when the Korotkoff sounds disappeared.

At least two readings were taken, with a minute between readings. Where readings differed by more than 4mm, more readings were taken until two similar consecutive readings were obtained. The lowest systolic reading, to the nearest two millimetres, was recorded with its corresponding diastolic reading.

Figure 3.5 Measurement of blood pressure using a paediatric size cuff.

7. Step test

The Queens College Step Test was used to assess physical fitness. This test proved to be accurate in the assessment of physical fitness in children as well as the estimation of VO_2max (Fardy *et al.*, 2000). $\dot{V}O_2max$ was calculated using the formula in Appendix G.

Although it is widely accepted that cycle ergometer tests have a higher correlation with directed measurements of $\dot{V}O_2max$, this test was used due to practical advantages.

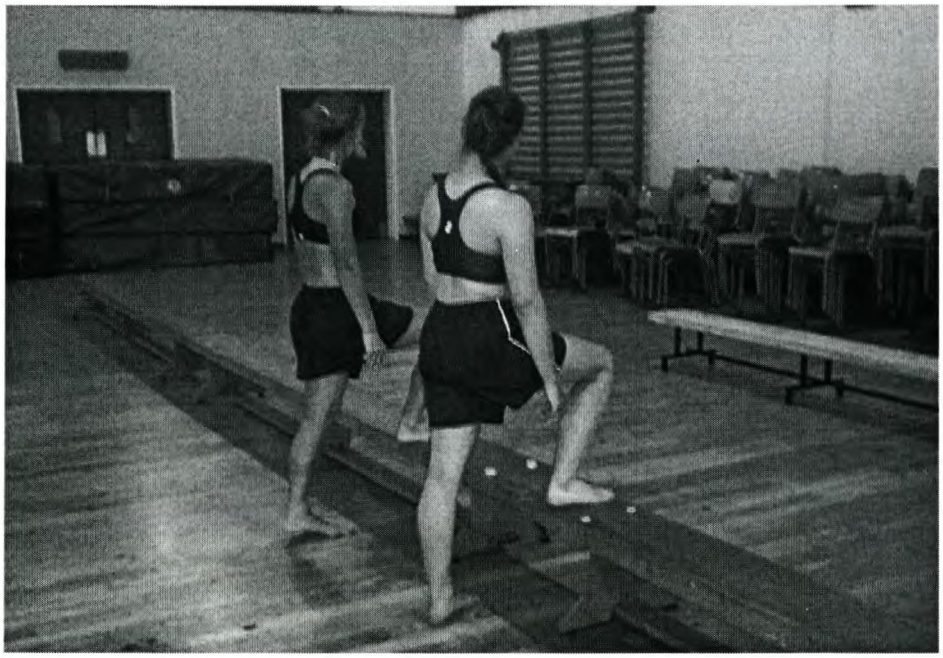

Figure 3.6 Subjects performing the step test.

8. Diet

Fat, sugar and salt intake was assessed with the use of a questionnaire completed by the parents (Appendix C). The questionnaire consisted out of 15 items that included high fat, high sugar and high salt food and beverages. For each item the parent gave the child a score of *one* to *four*, depending on how often the child consume that particular food or beverage. *One* indicated that the child rarely of never consumed that particular food or beverage and *four* indicated daily consumption.

Scores were added and each child received a score out of 60. Scores higher than 35 indicated that dietary intake can be classified as a risk factor for that particular child.

9. Family history

Family history was also assessed with a questionnaire (Appendix C) that was completed by the parents. The questionnaire was adapted from a questionnaire used by the American Academy of Family Physicians (Washington, 1999). This questionnaire, suitable for the assessment of family history, incorporated all aspects of family history that could lead to an increased risk for CAD, as defined by the American College of Sports Medicine (Kenney and Humphrey, 1995:18).

10. Activity levels

Activity levels were assessed with the use of two questionnaires, one that was completed by the parents and one that was completed by the subjects with the help of an assistant (Appendix C). These questionnaires concentrated on the participation of sports and activities, ways and means by which the child goes to and from the school, and the time the child spends on sedentary activity.

Data analysis

The Centre for Statistical Consultation, at Stellenbosch University, assisted in analysing the data. The Statistica 6.0 program was used.

Regression analyses were used in comparing variables and the influence it had on one another. A detailed description of the method used is provided in Appendix F.

CHAPTER IV RESULTS AND DISCUSSION

By reviewing the literature it is clear that risk factors for CAD are present among young children. There is thus reason to believe that the identification of CAD risk factors in small children can reduce the prevalence of CAD in the population. Early identification can lead to early intervention, which will have a positive influence on the morbidity and mortality of CAD. Results of this current study are compared to results in other similar studies.

Variable analysis

Frequency histograms are included for all the variables tested. On the frequency histograms one can clearly see what the frequency distribution of the tested variables is.

Box plots are included for certain variables. Medians, 25th and 75th percentiles, ranges and outliers can be identified on these plots. Medians are indicated by the small square (□). The bottom and top lines of the large square indicate the 25th and 75th percentiles, respectively. The vertical bars (whiskers) on either sides of the square indicate the range of the data. The outliers are represented by a small circle (°).

Pearson correlations were calculated between certain variables. Graphs of these correlations are also included.

ANOVA graphs of certain variables are included to demonstrate statistically significant differences between groups of children. The means of the data, in these graphs, are illustrated by a small circle (°). The vertical bars on either side of these circles illustrate the confidence intervals. Thus, a confidence interval of 0.95 indicates that it is 95% certain that the mean falls in the range illustrated by the vertical bars.

1. Age

The subjects' ages varied form 10.58 years to 14.58 years. The mean age of the boys tested was 12.35 years (SD = 0.92 years). The mean age of the girls tested was 12.35 years (SD = 0.89 years).

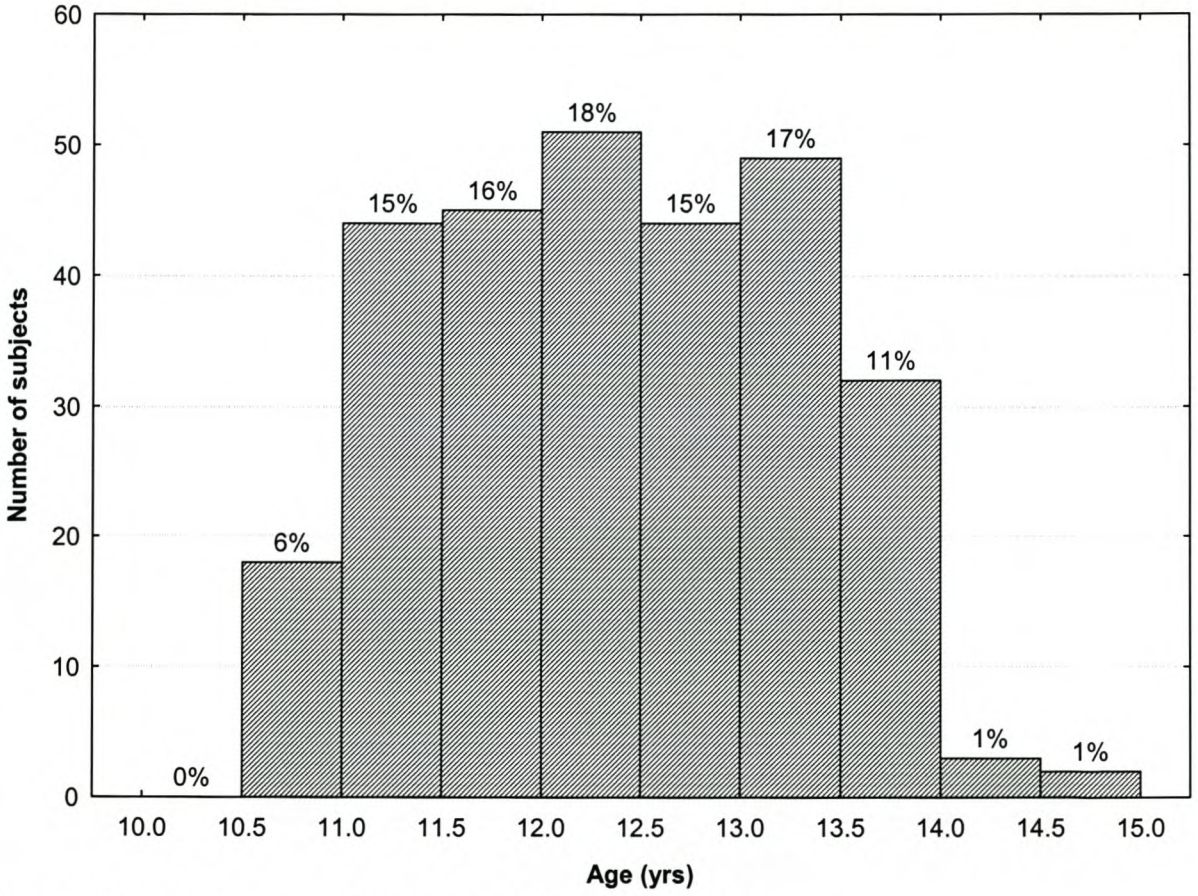

Figure 4.1 A frequency histogram of the sample's age.

2. Gender

Out of the 288 subjects tested 134 (47%) were girls and 154 (53%) were boys (Figure 4.2).

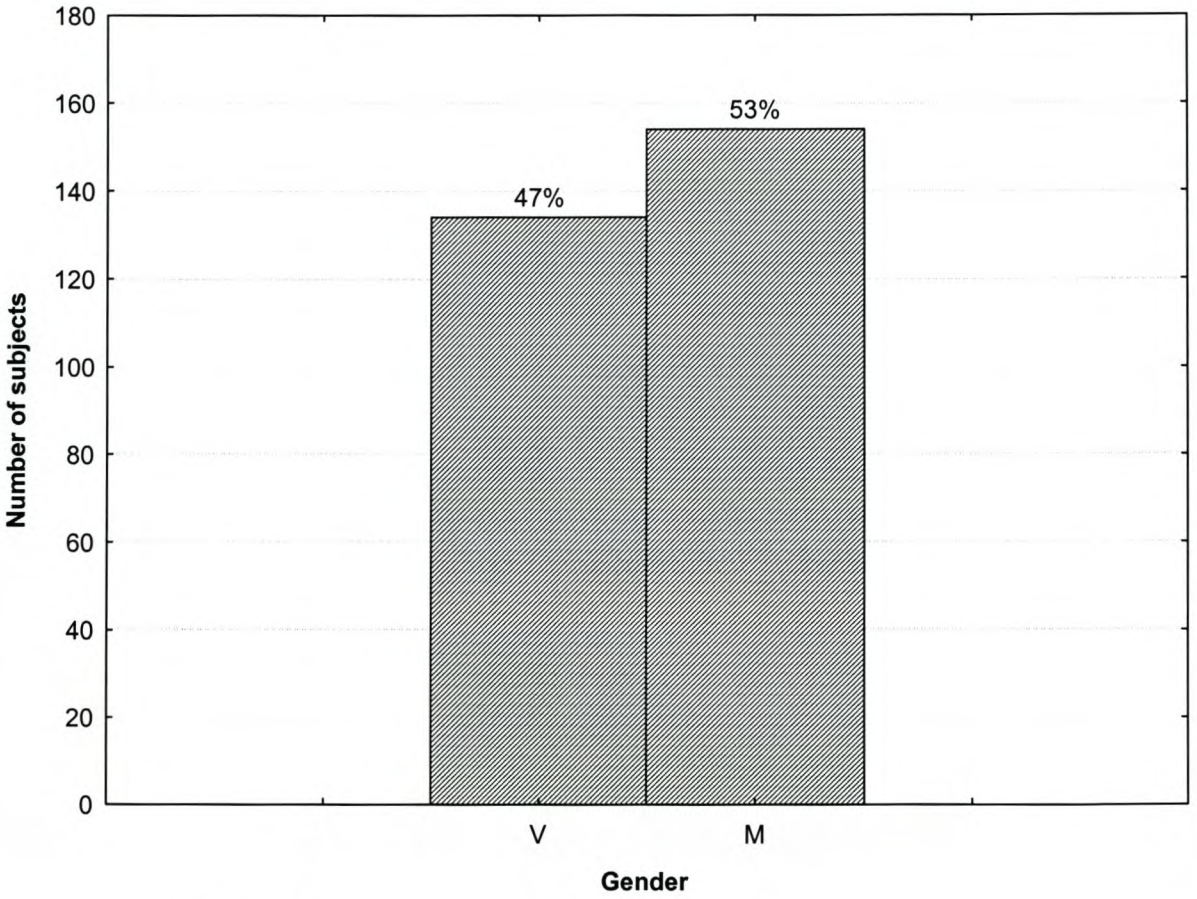

Figure 4.2 A frequency histogram of the sample's gender split.

3. Stature

The subjects' statures ranged from 1.29m to 1.83m (Figure 4.3). The mean height of the subjects was 1.55m (SD = 0.09m). The heights of the subjects were symmetrically distributed. The mean height of the girls was 1.54m (SD = 0.08m). The mean height of the boys was 1.55m (SD = 0.10m).

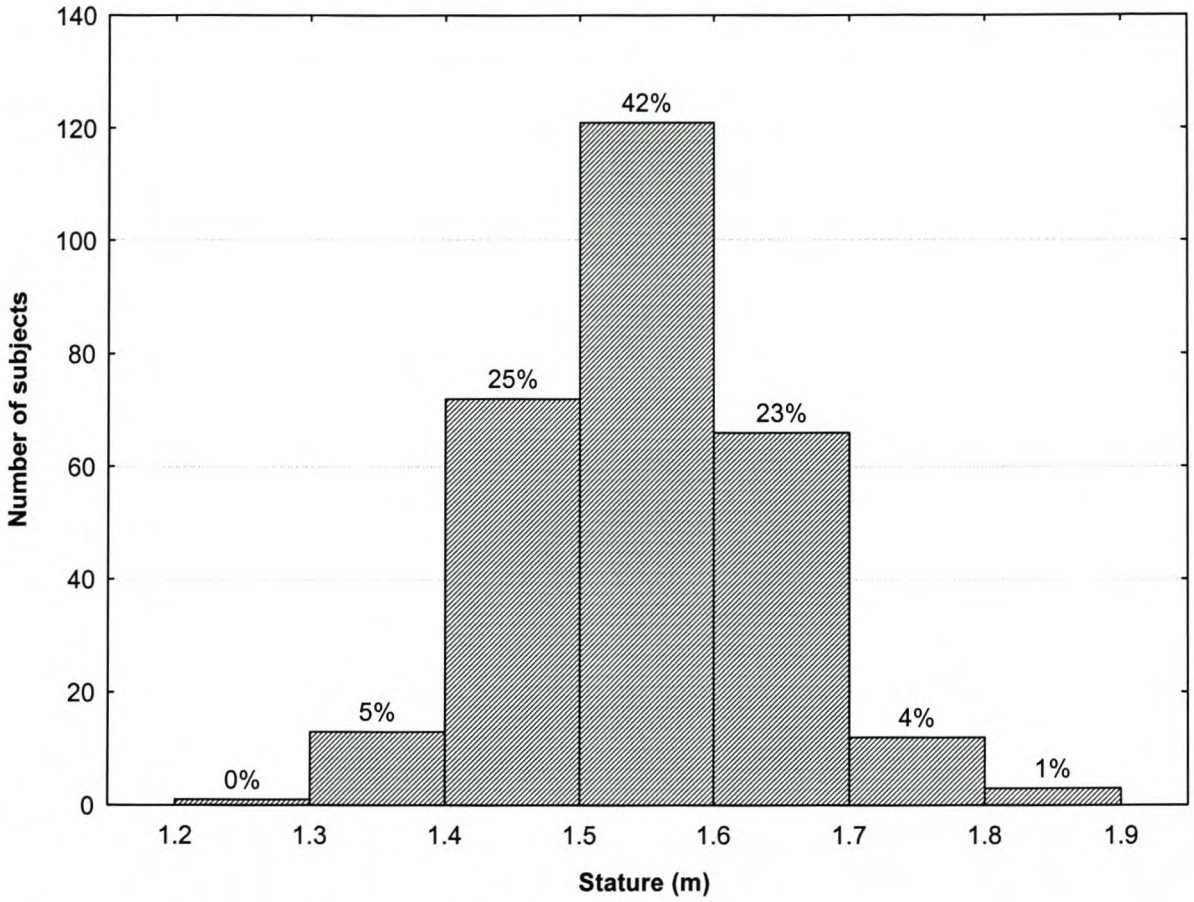

Figure 4.3 A frequency histogram of stature of subjects.

The hypothesis is that the mean statures of children tend to increase over the years, i.e. 11-year olds today are taller than 11-year olds were ten years ago. To confirm this hypothesis, one needs to test the children now and do a re-test in ten years time. By looking at other similar studies, one cannot statistically confirm this hypothesis, but by comparing the results a trend can be established. According to The Canadian Fitness Survey (Shepard, 1986) there was an increase in the stature of Canadian children over the years (Figure 4.4).

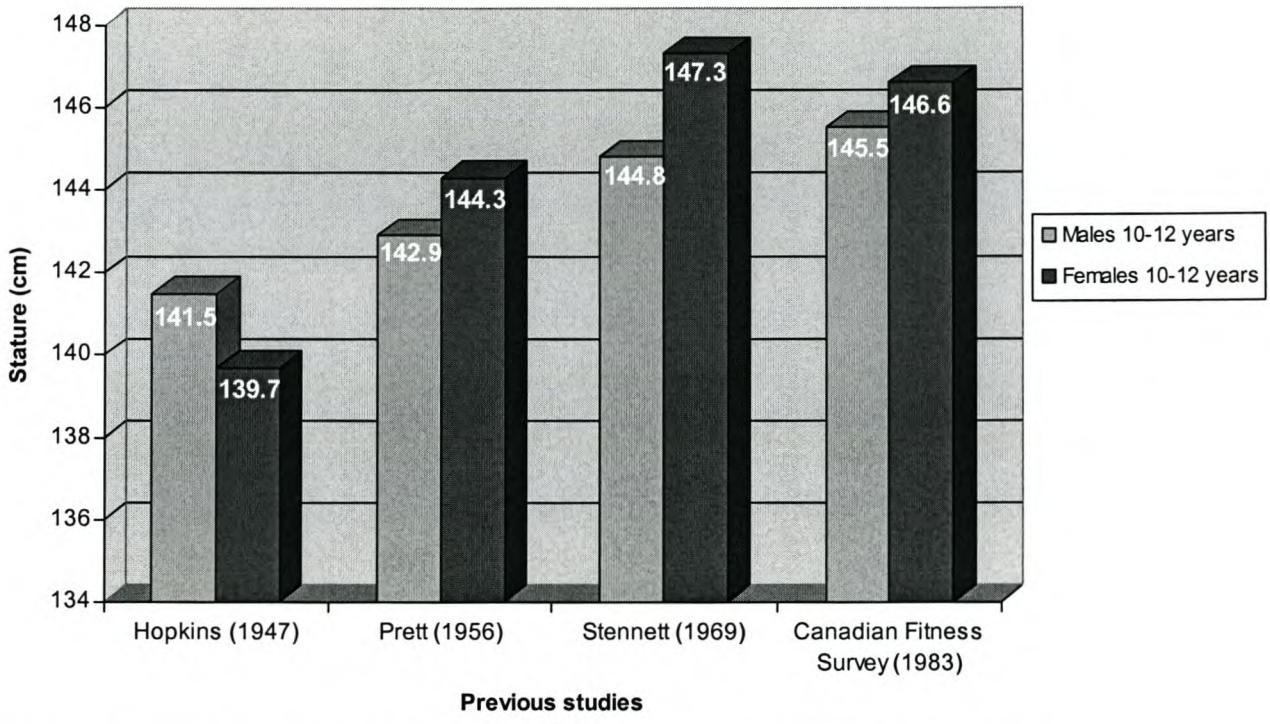

Figure 4.4 A graphic illustration of the secular trend of the increase of stature in Canadian children (Shepard, 1986).

The findings of other studies on the stature of children are presented in table 4.1. No trend can be established, probably because of the age and geographic differences of the subjects.

Table 4.1 The stature of children as found in other studies

	Girls		Boys	
	Stature (cm)	**Age (years)**	**Stature (cm)**	**Age (years)**
Jetté *et al.* (1984)	157.9 (SD = 5.9)	12.9 (SD = 0.62)	156.6 (SD = 8.7)	12.5 (SD = 0.83)
Rossouw (1990:LVII)	145.0 (SD = 7)	11	143.0 (SD = 5)	11
Boreham *et al.* (2001)	151.0 (SD = 7.5)	12	149.7 (SD ± 7.9)	12
Current study	154.0 (SD = 8)	12.35 (SD = 0.89)	155.0 (SD = 10)	12.35 (SD = 0.92)

The study by Jetté and associates (1984) was conducted by the University of Ottawa and the University of Waterloo on 11- to 14-year-old children. The mean age of the children was the same as the mean age of the children in the current study. The study by Rossouw (1990) was done in the same geographic location as the current study. The mean age of the subjects was just younger than the subjects tested in the current study. The study by Boreham and associates (2001) was done on Irish 12-year olds.

When looking at the norms above and the norms in figure 4.4, it is clear that the stature of girls is taller in all the age groups. In this current study's data the boys were on average taller than the girls. Even when looking at the study by Rossouw (1990:LVII), in South Africa in the Western Cape, the girls were on average taller.

The median for the stature of boys ages 11 to 13, according to Martin and Ward (1996:114-116), is 150.867cm. The median for the stature of the boys in the current study is 155cm.

The median for the stature of girls ages 11 to 13, according to Martin and Ward (1996:121-123), is 152.367cm. The median for the stature of the girls in the current study is 155cm. When looking at the medians for children's stature, according to Martin

and Ward (1996:123-144), the median for the girls was again higher than that of the boys. When looking at the data from the current study, the medians for the boys and the girls were exactly the same.

The boys' mean stature is higher than the mean stature of the girls. There were however, three outliers in the boys' stature above 1.8m (Appendix E). When calculating the boys' mean stature without the three outliers, the boys' mean stature is 154.3cm. There is no significant difference between the mean stature of the girls and the boys, when the boys' mean stature is calculated without the outliers. The medians and the ranges of the boys' and girls' stature are very similar, confirming that the outliers are the cause for the higher mean of the boys (Appendix E).

4. Body weight

Body weight of the subjects ranged from 24.9kg to 88.4kg (Figure 4.5). The subjects' mean weight was 47.5kg (SD = 11.20kg). The mean weight of the girls was 49.13kg (SD = 11.18kg). The mean weight of the boys was 46.09kg (SD = 11.05kg).

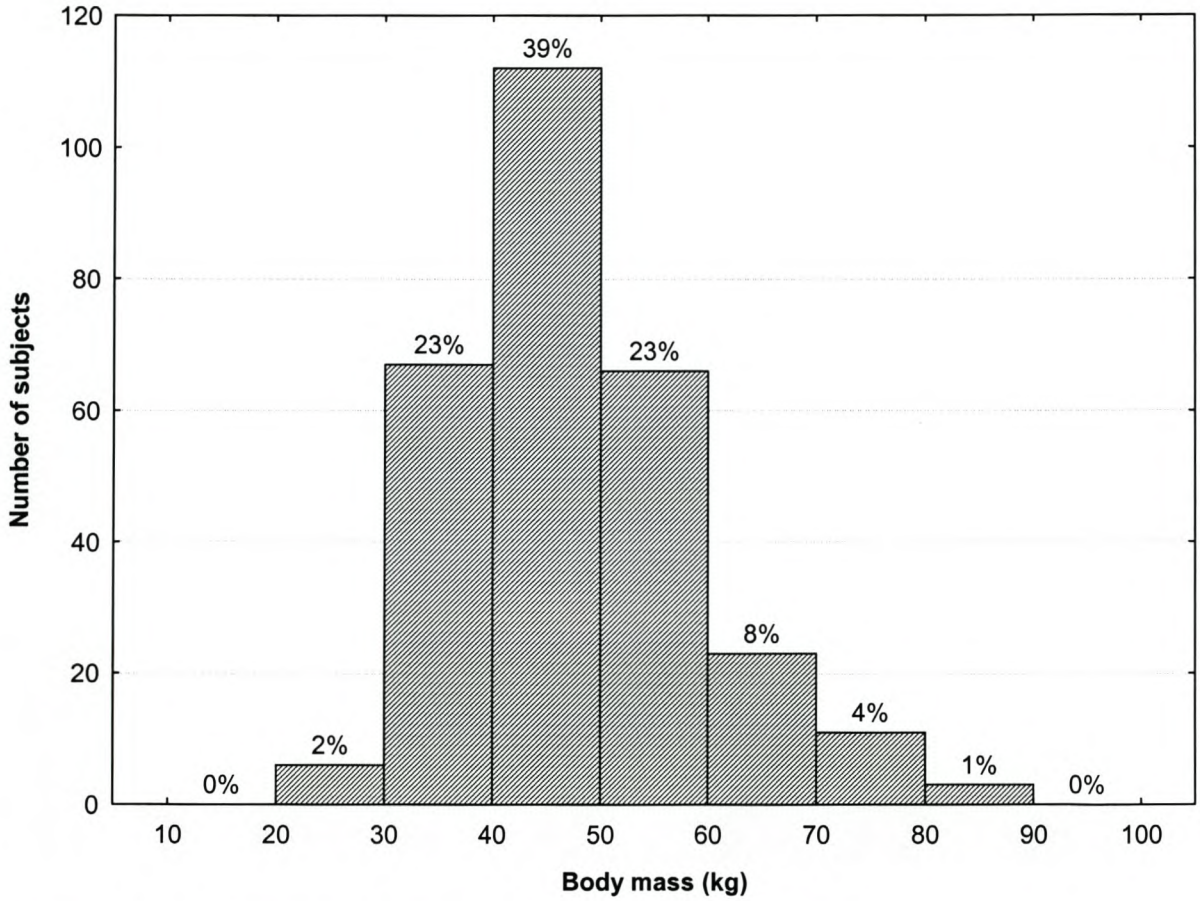

Figure 4.5 A frequency histogram of body weight of subjects.

Findings on the body weight of children, in more or less the same age group as this study, done by other researchers are presented in Table 4.2.

Table 4.2 Body weight of children, as found by other researchers.

	Girls		Boys	
	Weight (kg)	**Age (years)**	**Weight (kg)**	**Age (years)**
Jette *et al.* (1984)	46.4 (SD = 6.6)	12.9 (SD = 0.62)	43.8 (SD = 8.6)	12.5 (SD = 0.83)
Rossouw (1990:LVIII)	37.5 (SD = 7.4)	11	36.4 (SD = 7.0)	11
Boreham *et al.* (2001)	44.0 (SD = 9.0)	12	42.6 (SD = 9.4)	12
Current study	49.14kg (SD = 11.18kg)	12.35 (SD = 0.89)	46.09kg (SD = 11.05kg)	12.35 (SD = 0.92)

In this current study, the body weight of the boys and the body weight of the girls were higher than those mentioned in the studies above. The study initiated by Boreham and associates (2001) was on Irish children and the attributes of the subjects were very similar to the sample used in this study. The study by Rossouw (1990) was also on South African children. The big difference in body weight in that study compared to the current study, can possibly be ascribed to the fact that the mean age of the subjects in that study was lower than the mean age of the subjects in the current study. According to The Canadian Fitness Survey (Shepard, 1986), the mean body weight for a 10-year-old should be 30.8kg for boys and 30.4kg for girls. The mean body weight for a 12-year-old should be 37.4kg for boys and 39.0kg for girls (Shepard, 1986).

According to Martin and Ward (1996:114-116) the median for the body weight of boys ages 11 to 13 is 41.8kg. The median for the boys' body weight in the current study was 44.8kg.

The median for body weight of girls ages 11 to 13 is 43.63kg (Martin and Ward, 1996:121-123). The median for the girls' body weight in the current study was 47.7kg.

Here it is clear that the body weight of the children tested in this study was more than what the norms suggest ($p<0.001$).

5. Body Mass Index (BMI)

The BMI of the subjects ranged from 13.84kg/m^2 to 35.51kg/m^2 (Figure 4.6). The subjects' mean BMI was 19.75kg/m^2 (SD = 3.69kg/m^2). The mean BMI of the girls was 20.54kg/m^2 (SD = 3.87kg/m^2). The mean BMI of the boys was 19.06kg/m^2 (SD = 3.40kg/m^2).

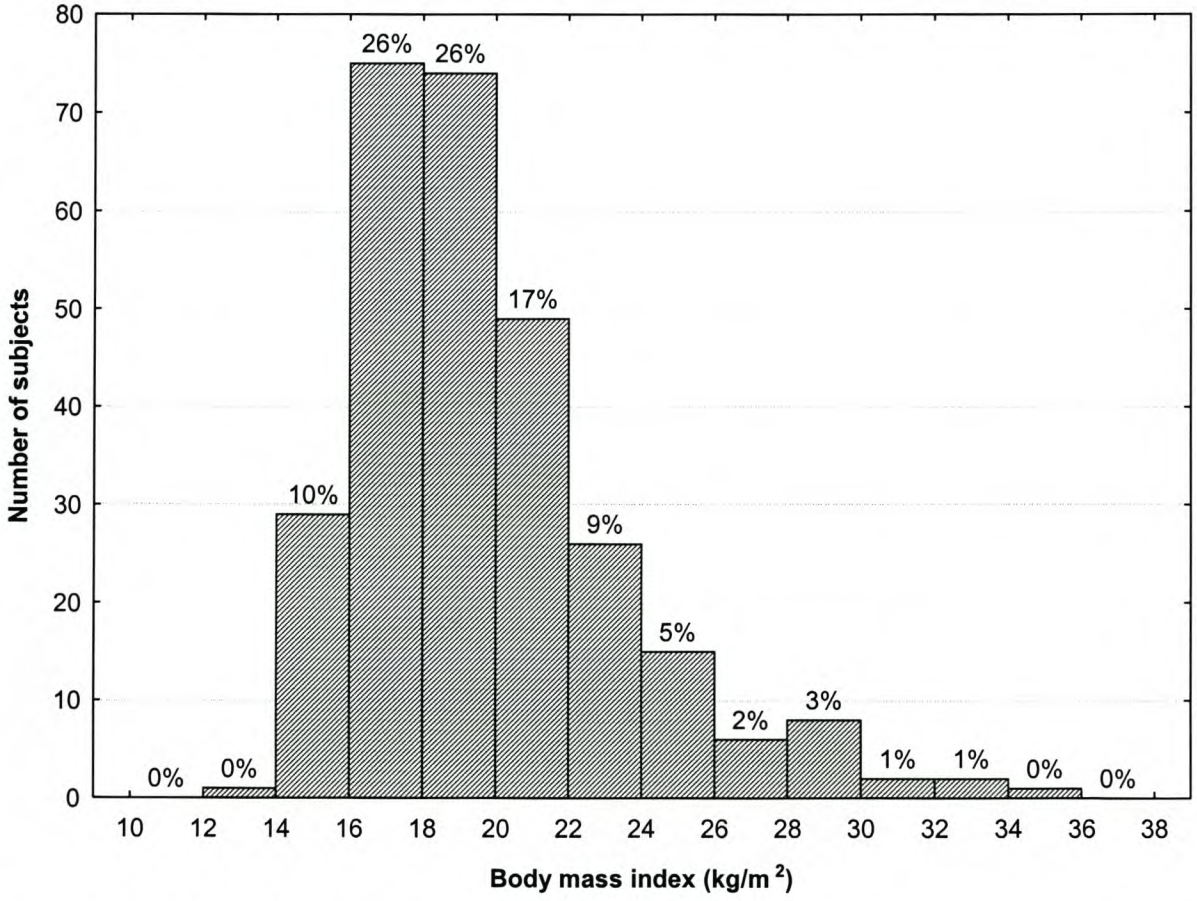

Figure 4.6 A frequency histogram of subject's body mass index.

As seen in Figure 4.6, more than half (52%) of the subjects tested had a BMI of between 16kg/m^2 and 20kg/m^2. The norms most commonly used for the classification of BMI

were developed for adults. Whether these norms are applicable to children are not yet established. The desirable value for the BMI of adults is at or below 24kg/m^2. Eighty eight percent of the children had a BMI at or below this value. However, in the study done by Rossouw (1990:LIX) the mean BMI for the girls was 17.8kg/m^2 (SD = 2.4kg/m^2) and the mean BMI for the boys was 17.7kg/m^2 (SD = 2.7kg/m^2). The mean BMI of the girls and the boys tested in the current study are clearly higher than those tested by Rossouw (1990:LIX).

6. Waist-Hip Ratio

The waist-hip ratios of the subjects ranged from 0.50 to 0.99 (Figure 4.7). The subjects' mean waist-hip ratio was 0.78 (SD = 0.05). The mean waist-hip ratio of the girls was 0.78 (SD = 0.22). The mean waist-hip ratio of the boys was 0.81 (SD = 0.04).

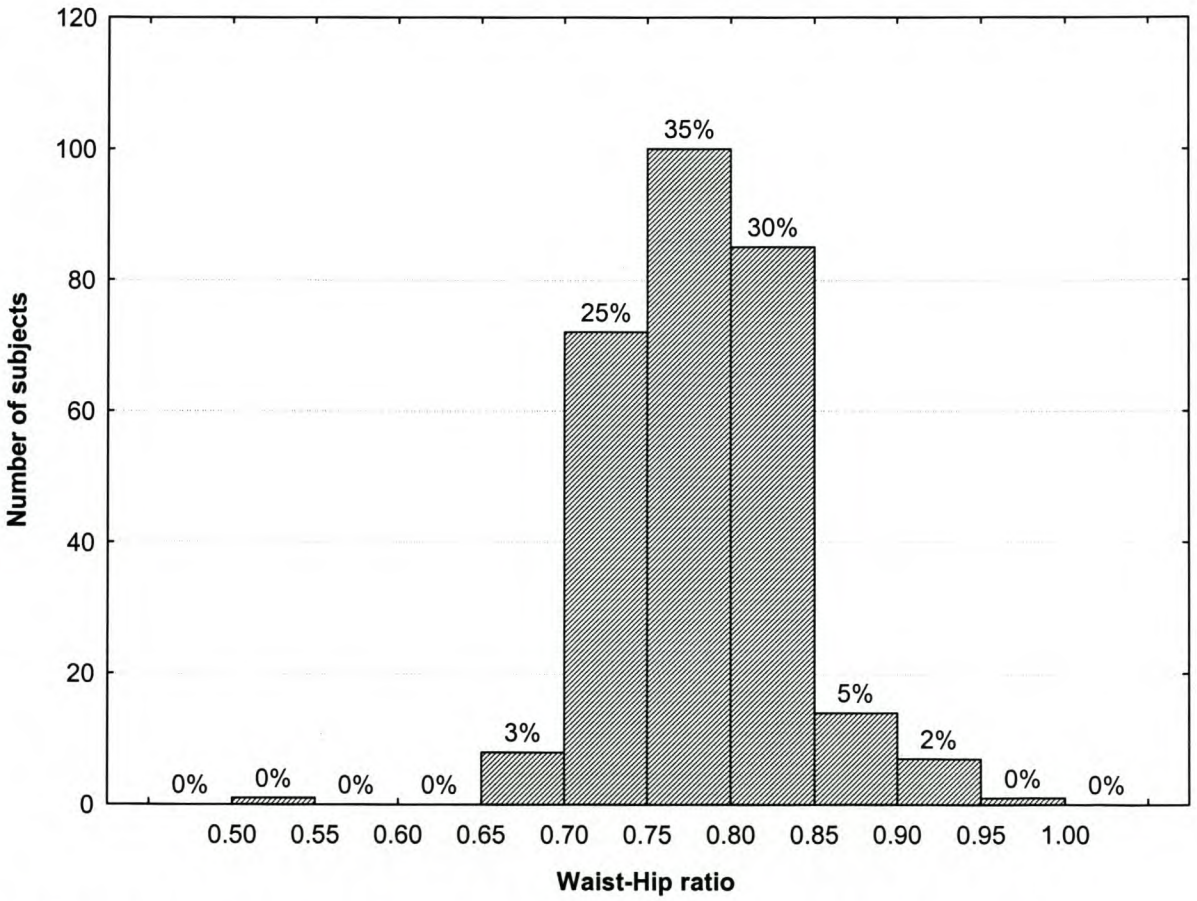

Figure 4.7 A frequency histogram of the subjects' waist-hip ratios.

The norms used for waist-hip ratio calculations, are the same norms used for adults (significant health risk at >0.95 for men and >0.8 for women). Whether these norms are suitable for children is still debatable. Norms for waist- and hip circumferences are presented in Table 4.3.

Table 4.3 Norms for waist- and hip circumferences (Crawford, 1996:70-71).

	Boys		**Girls**	
	Waist (cm)	**Hip (cm)**	**Waist (cm)**	**Hip (cm)**
11-year-old	63.96 (SD = 6.30)	75.63 (SD = 6.70)	61.31 (SD = 6.50)	77.12 (SD = 7.27)
12-year-old	65.39 (SD = 6.15)	78.12 (SD = 6.52)	63.67 (SD = 6.50)	82.15 (SD = 7.19)
13-year-old	67.65 (SD = 6.23)	81.82 (SD = 7.17)	66.20 (SD = 6.90)	86.98 (SD = 7.53)

If one calculates the waist-hip ratio of the above norms it ranges from 0.83-0.85 for boys and 0.76-0.79 for girls. When comparing these to the norms used for adults (p. 17), it is noticeably lower. In the light of this it is understandable that some (James, 2001; Kalk, 2001) suggest the use of only the waist circumference for children. In the current study the mean waist-hip ratio for the girls was 0.78 (SD = 0.22) and for the boys 0.81 (SD = 0.04). These results both fall below the norms used for adults. If compared to Crawford's (1996:70-71) norms, the mean waist-hip ratio for the boys fall below the desirable range and the girls fall within that range. Looking at the waist circumference alone, the mean circumference for the boys and girls was 66.27cm (SD = 8.10cm) and 66.05 (SD = 8.29cm), respectively. These results are not as favourable as the results when calculating waist-hip ratio. When calculating the mean waist circumference for children 11 to 13 years from Crawford's norms, the mean for boys is 65.67cm and 63.73cm for girls. Taking in account the standard deviation, the results for the girls and the boys are considerably higher than the mean indicated by Crawford (1996:70-71).

7. Tricep skinfold

The tricep skinfolds of the subjects ranged from 4.2mm to 38.4mm (Figure 4.8). The subjects' mean tricep skinfold was 13.96mm (SD = 6.98mm). The mean tricep skinfold of the girls was 13.96mm (SD = 6.98mm). The mean tricep skinfold of the boys was 11.97mm (SD = 6.25mm).

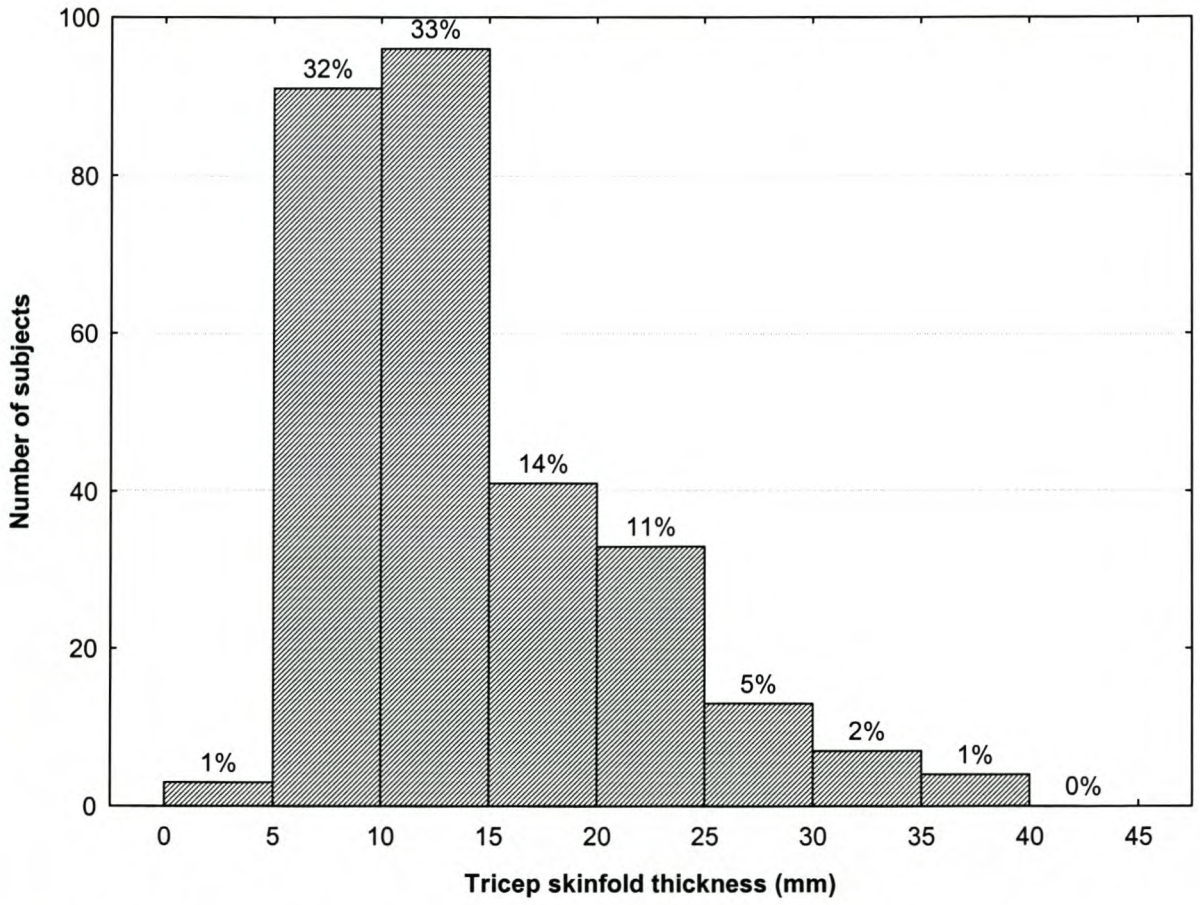

Figure 4.8 A frequency histogram of the subjects' tricep skinfolds.

The tricep skinfold of 66% of the subjects tested was 15mm or less. According to Martin and Ward (1996:114-123) the median for boys ages 11 to 13 is 9.57mm. The median for girls in this age group is 12.43mm. The median tricep skinfold thickness for the girls tested was 13.8mm, which is slightly higher than the median of the norms mentioned above. The median tricep skinfold thickness for the boys tested was 10.2mm, which is

also higher than the norms mentioned above. The most cumbersome attribute about this data is the wide range of skinfold thickness. Very high deposits of subcutaneous fat (>20%) were present in 19% of the subjects.

8. Subscapular skinfold

The subscapular skinfolds of the subjects ranged from 3.2mm to 42mm (Figure 4.9). The subjects' mean subscapular skinfold was 11.02mm (SD = 7.86mm). The mean subscapular skinfold of the girls was 13.36mm (SD = 8.01mm). The mean subscapular skinfold of the boys was 8.98mm (SD = 7.15mm).

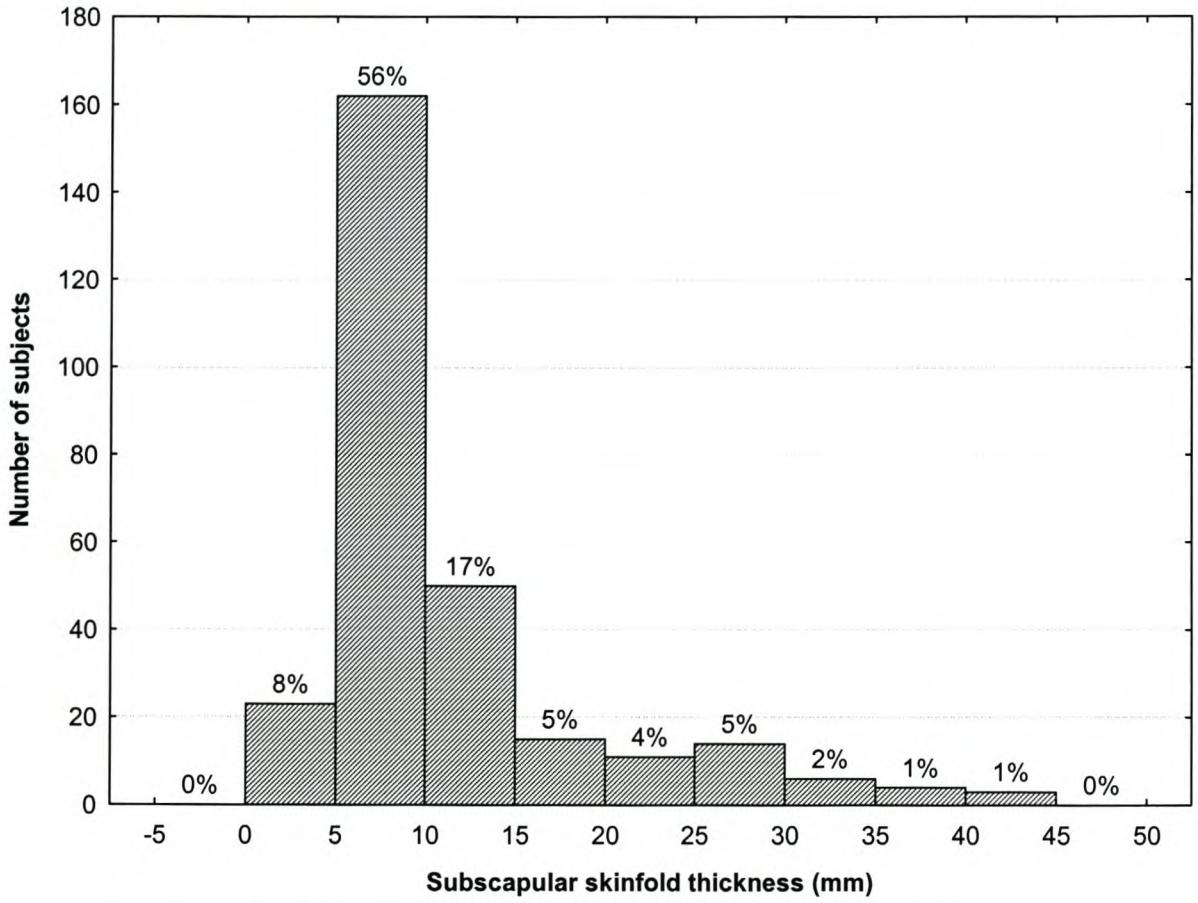

Figure 4.9 A frequency histogram of subjects' subscapular skinfolds.

The subscapular skinfold of 64% of the subjects tested was 10mm and lower. The median for boys in this age group is 6.17mm. The median for girls in this age group is 8.03mm (Martin and Ward, 1996:114-123). The median subscapular skinfold thickness for the girls tested was 10.4mm, which is well above the median of the norms mentioned above. The median tricep skinfold thickness for the boys tested was 6.6mm, which is not noticeably higher than the norms mentioned above.

9. Fat percentage

The fat percentages of the subjects ranged from 8.21% to 77.77% (Figure 4.10). The subjects' mean percentage body fat was 21.99% (SD = 10.05%). The percentage body fat for girls ranged from 12.19% to 50.76%; their mean percentage body fat was 24.66% (SD = 8.92%). The mean percentage body fat of the boys was 19.67% (SD = 10.42%).

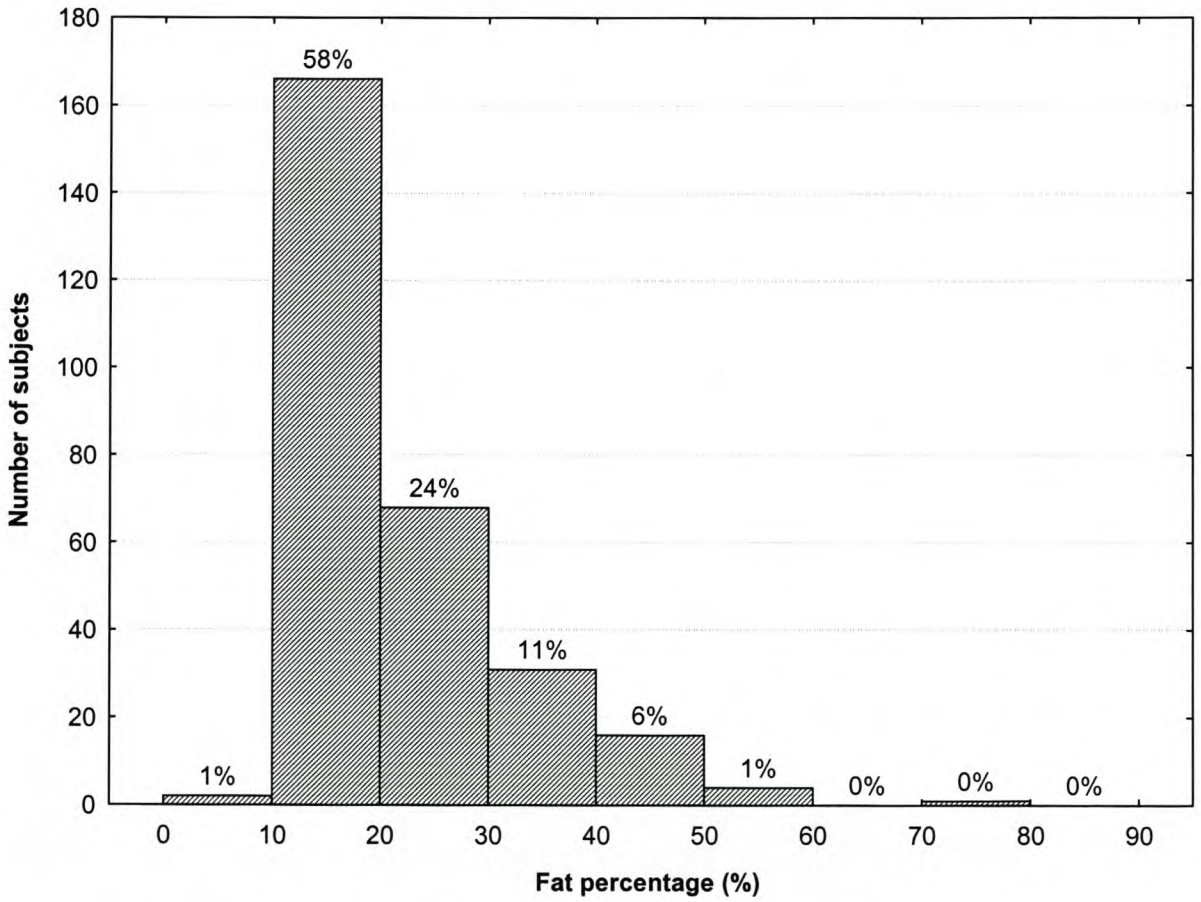

Figure 4.10 A frequency histogram of subjects' fat percentage.

The percentage body fat of 59% of the subjects tested was 20% and lower. In a study done by Sleap and Tolfrey (2001) on nine to twelve year old children, the mean percentage body fat for the girls and the boys were 18.9% (SD = 6.5%) and 15.7% (SD =± 5.4%), respectively. In this study the same skinfold sites and equations were used to estimate percentage body fat, as in this current study. Both the girls and the boys tested had a higher mean percentage body fat, 24.66% (SD = 8.92%) and 19.67% (SD = 10.42%) for the girls and boys respectively, than those tested by Sleap and Tolfrey (2001).

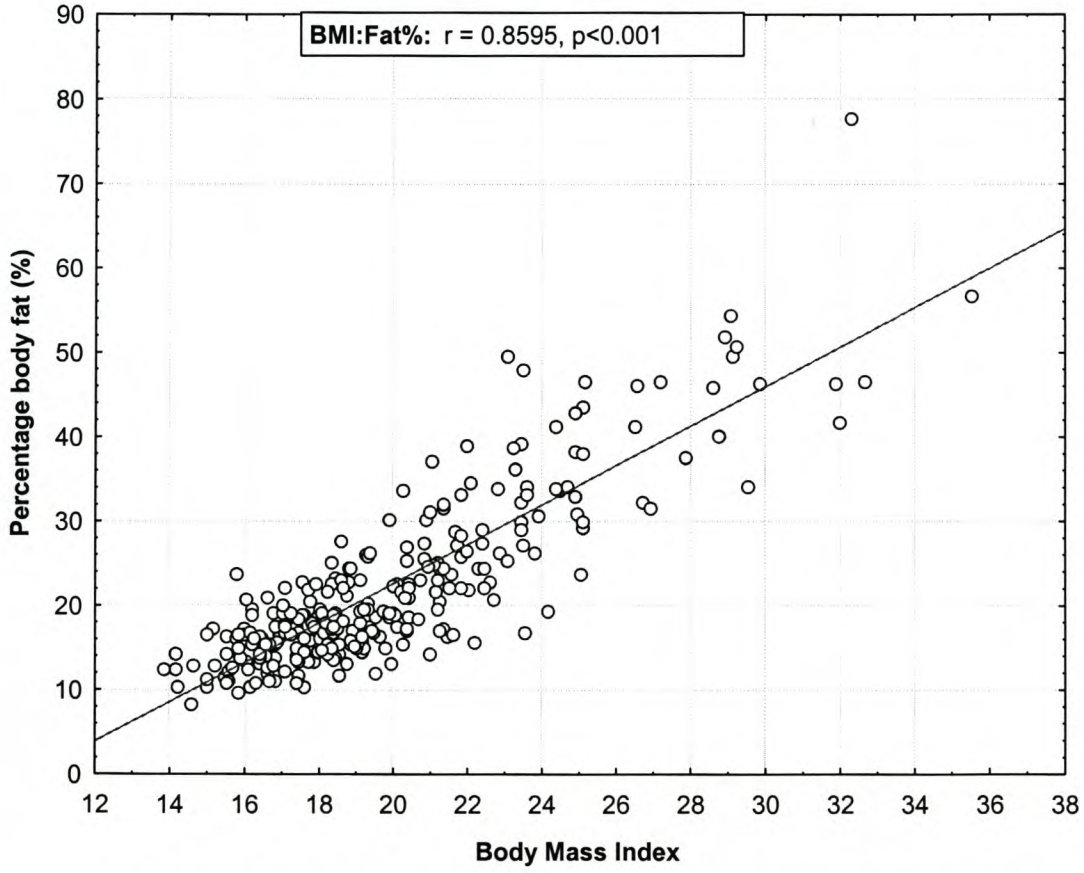

Figure 4.11 A scatter plot to illustrate the correlation between percentage body fat and body mass index.

As seen in figure 4.11, the correlation between percentage body fat and BMI of the subjects was 0.8595 (p<0.001). Thus, when it is not possible to measure percentage

body fat, BMI can be used to give an indication of what the influence of a child's body composition is on his/her CAD risk. Keep in mind that BMI does not differentiate between fat- and muscle mass.

10. Activity levels

The activity scores of the subjects ranged from 1 to 15 (Figure 4.12). The mean for the subjects' activity scores was 8.48 (SD = 3.15). The activity scores for girls ranged from 1 to 14; the mean for their activity scores was 8.04 (SD = 3.4). The mean for the boys' activity scores was 8.86 (SD = 2.86); their scores ranged from 2 to 15.

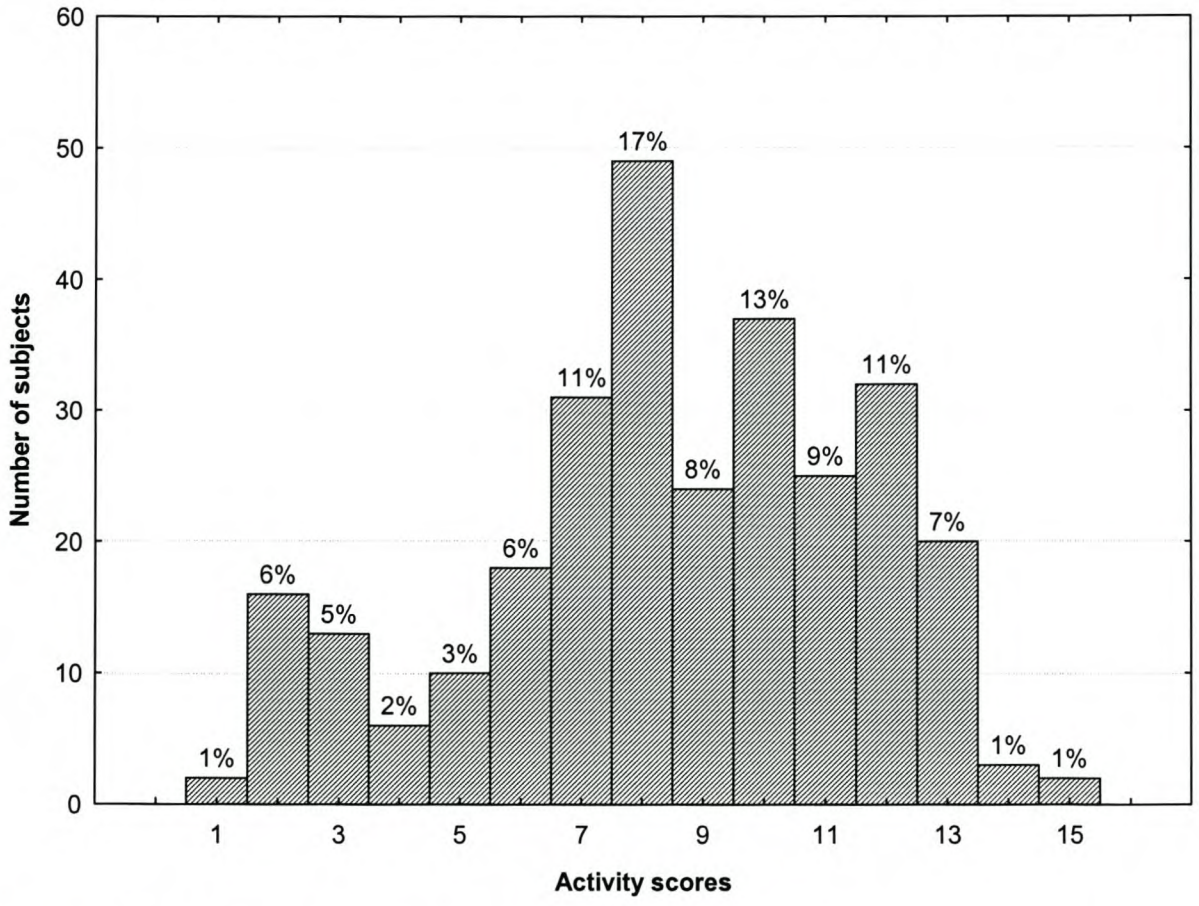

Figure 4.12 A frequency histogram of subjects' activity scores.

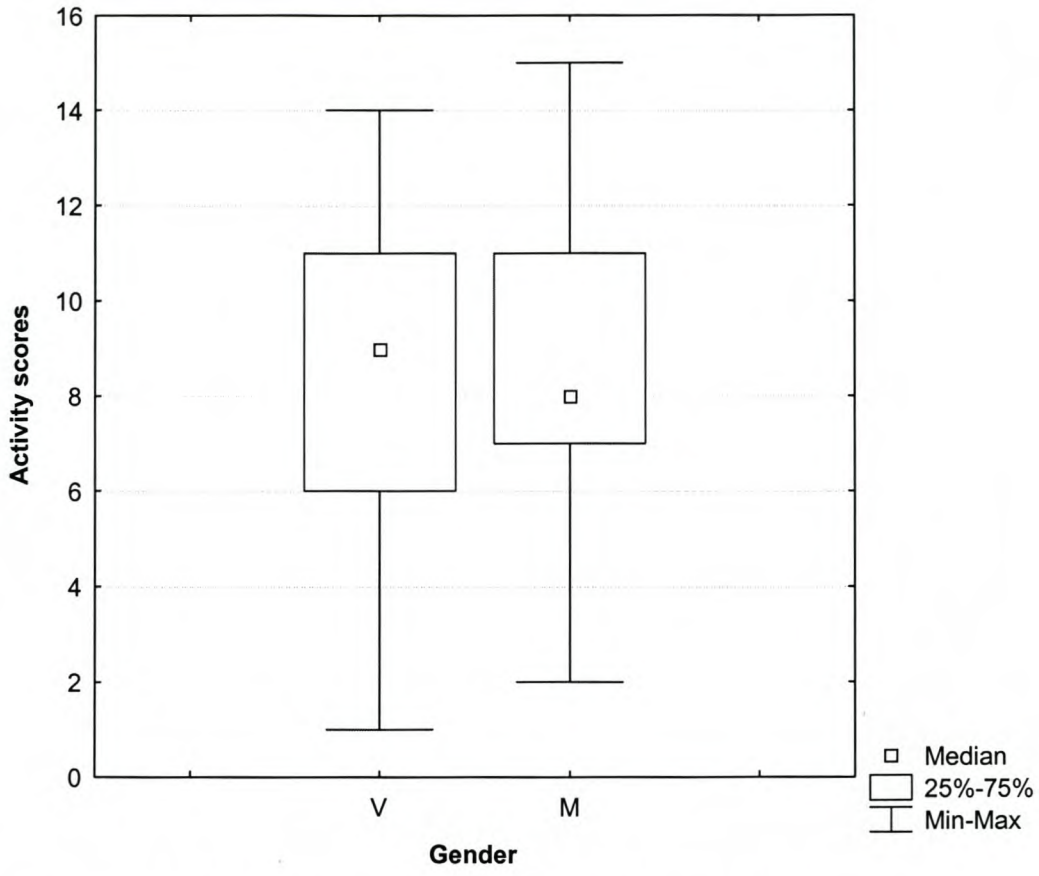

Figure 4.13 A box plot illustrating the activity scores of the subjects.

There was a significant difference between the activity scores of the boys and the girls (p=0.02). The activity scores for the girls ranged from one to 14 (Figure 4.13). The median for the girls is 9. The 25th and 75th percentile is six and 11, respectively. The activity scores for the boys ranged form two to 15. The median for the boys is 8. The 25th and 75th percentile is seven and 11, respectively.

It was decided beforehand that activity scores of eight and above are considered as "active". Thus, subjects with these scores engage in sufficient physical activity for long-term health promotion. The activity score of 67% of all the subjects was eight and above. Thus, 33% of all the subjects tested did not engage in sufficient physical activity and were considered as inactive. Considering the age group of the subjects tested, one would have hoped for a higher percentage of active children. Most people assume that pre-pubescent children are naturally active. The fact that some of the subjects tested

had already reached puberty could have an influence on their activity levels, especially the girls. On the other hand, the fact the all the subjects were still in primary school should be beneficial for physical activity. Primary schools usually put a higher premium on physical education and participation in school-based sports. Of all the subjects, 30.52% of the boys and 36.57% of the girls are considered as inactive. There was a statistically significant difference between the activity levels of the girls and the boys ($p=0.02$).

11. Systolic blood pressure

The systolic blood pressure of the subjects ranged from 82mmHg to 160mmHg (Figure 4.14). The mean for the subjects' systolic blood pressure was 117.91mmHg (SD = 14.07mmHg). The mean systolic blood pressure for the girls was 117.91mmHg (SD = 14.42mmHg). The mean for the boys' systolic blood pressure was 117.92mmHg (SD = 13.81mmHg).

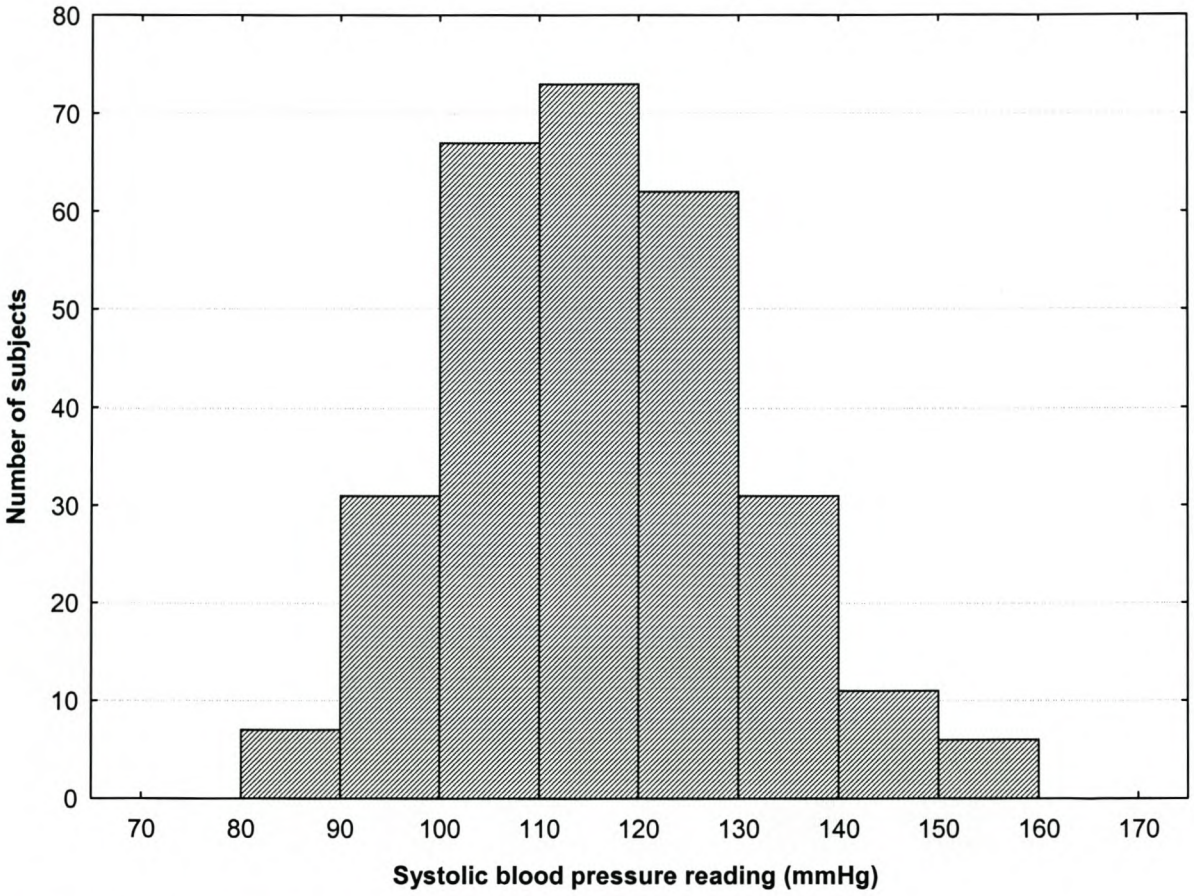

Figure 4.14 A frequency histogram of systolic blood pressure.

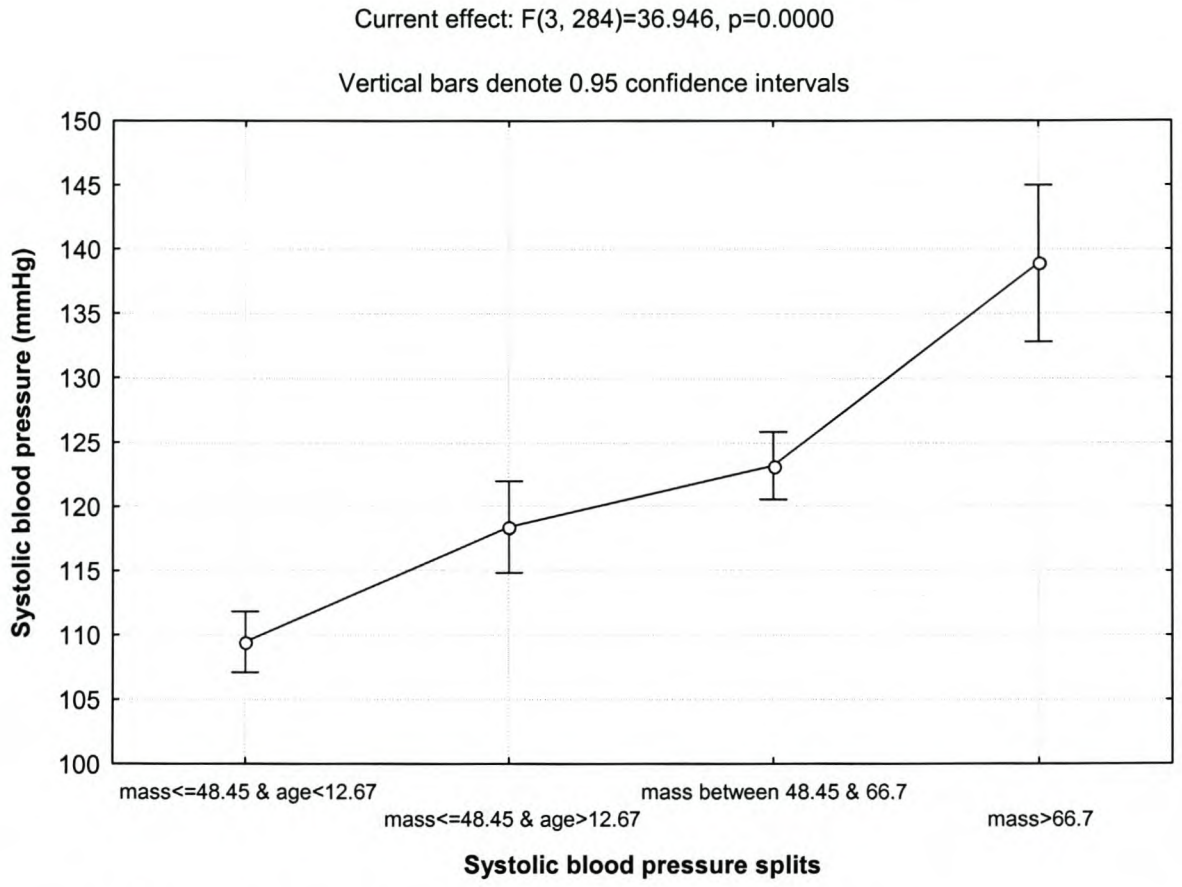

Figure 4.15 ANOVA graph illustrating systolic blood pressure splits.

Body mass and age were identified as the determents of systolic blood pressure (figure 4.15). A regression tree analysis was done to identify homogeneous groups of children with regards to systolic blood pressure readings (Appendix F). This analysis was done for the group as a whole. There was no significant difference between the systolic blood pressure of the girls and the systolic blood pressure of the boys (p=0.100). The mean systolic blood pressure reading of the subjects under the age of 12.67 and with a body mass lower or equal to 48.45kg was 109mmHg. The mean for the systolic blood pressure reading for subjects above the age of 12.67 and with a body mass lower or equal to 48.45kg was 118mmHg. The mean systolic blood pressure reading for subjects with a body mass between 48.45kg and 66.7kg was 124mmHg. There is one subject with a systolic blood pressure of 160mmHg in this group, it is seen as an outlier

(Appendix E). The mean systolic blood pressure reading for subjects with a body mass higher than 66.kg was 139mmHg.

There was a statistically significant difference between the systolic blood pressure readings of the first and second group ($p<0.001$). There was, however, no significant difference between the readings of the second and third group ($p=0.21$). The systolic blood pressure readings of the third and forth group were significantly different ($p<0.001$).

If one look at the first two groups, it appears that age seems to play a role in blood pressure readings. The body masses of these subjects were the same and still there were a statically significant difference in their systolic blood pressure readings. There is controversy about the age groupings of children and adolescents when it comes to the classification of blood pressure readings. Some authors consider 13-year olds children and some consider them adolescents. It seems however, that around 13 years there is a natural rise in systolic blood pressure when considering that the suggested systolic blood pressure readings for adolescents are noticeably higher than that of children. The difference in systolic blood pressure readings of the subjects older and younger than 12.67 years, respectively, can thus be due to natural causes.

The systolic blood pressure readings of the forth group are statistically significantly higher than that of the third group ($p<0.001$). This just confirms what research has shown; body mass does influence systolic blood pressure readings. This indicates that if a child in this age group has a body mass of 66.7kg or higher, there is a good chance that that child will have an elevated systolic blood pressure reading.

A child is classified with mild to moderate hypertension when blood pressure is above or equal to 130mmHg for systolic blood pressure or above or equal to 86mmHg for diastolic blood pressure. Severe hypertension is diagnosed when blood pressure is above or equal to 144mmHg for systolic blood pressure or above or equal to 96mmHg for diastolic blood pressure (Plowman and Smith, 1997:194).

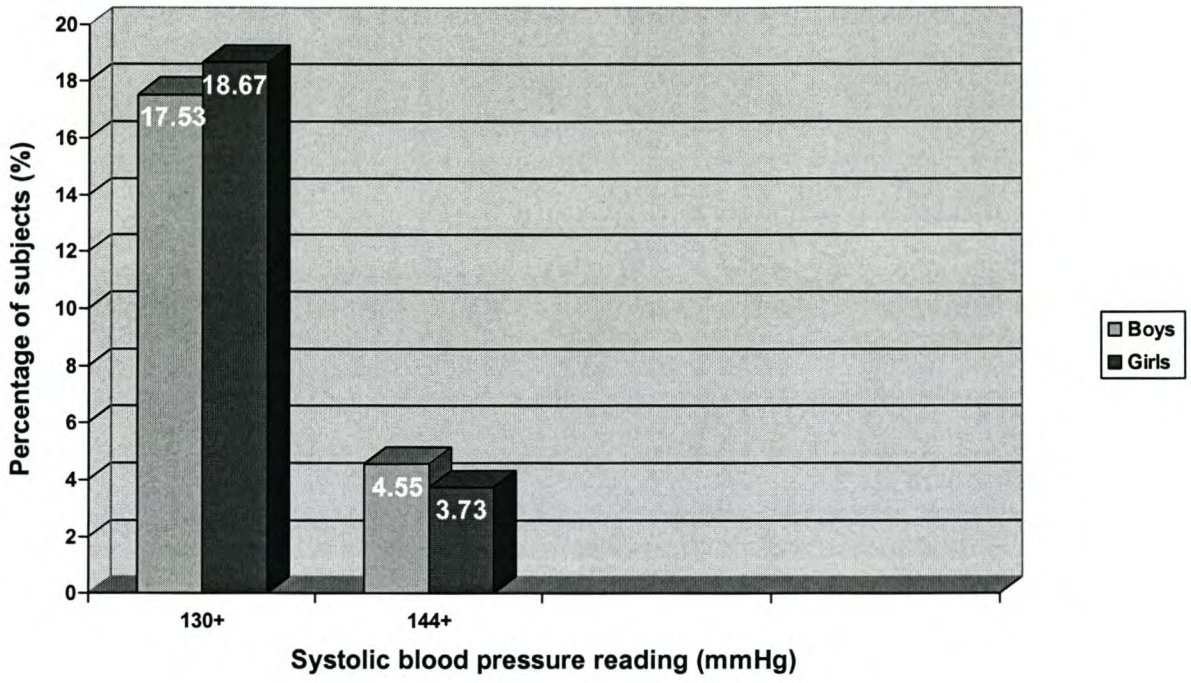

Figure 4.16 A graphic illustration of the percentage subjects with moderate and severe hypertension.

In figure 4.16 the percentage of boys and girls with moderate and severe hypertension, respectively, is illustrated. In other words, 18.06% of all the subjects tested had a systolic blood pressure reading indicating mild to moderate hypertension. Of all the subjects tested, 4.17% had a systolic blood pressure reading indicating severe hypertension.

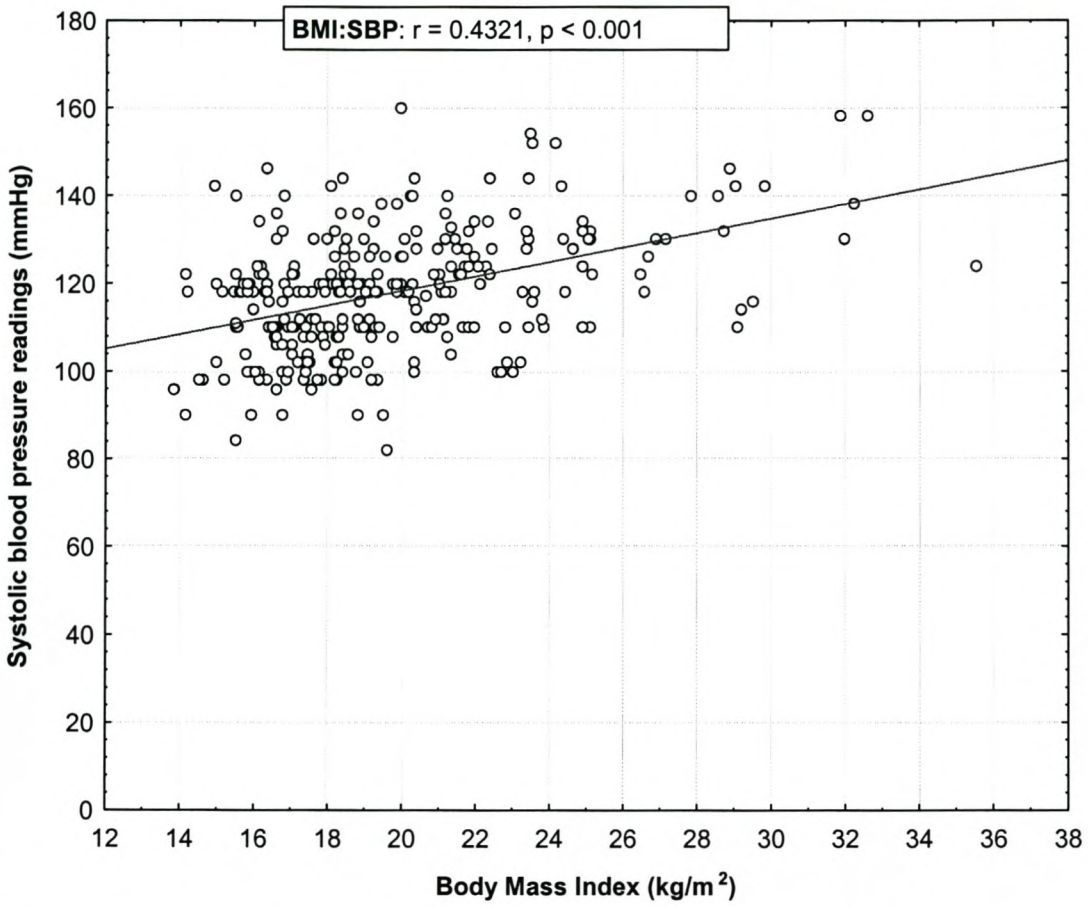

Figure 4.17 A scatter plot to illustrate the correlation between systolic blood pressure and body mass index.

Figure 4.17 illustrates the correlation between systolic blood pressure readings and BMI. The correlation between these two variables was 0.4321 ($p<0.001$). The correlation is not strong enough to assume that a child with a high BMI will have systolic hypertension.

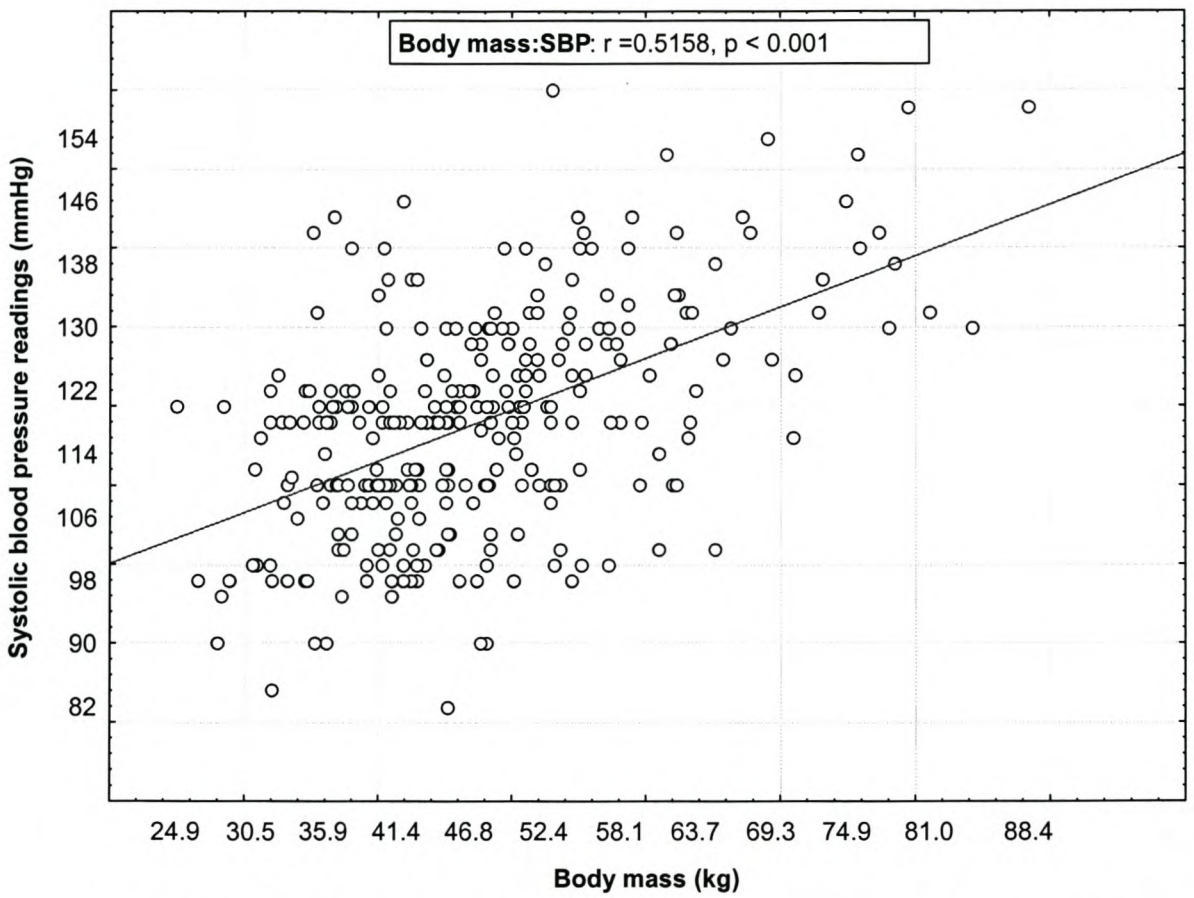

Figure 4.18 A scatter plot to illustrate the correlation between systolic blood pressure readings and body mass.

The correlation between systolic blood pressure and body mass is 0.5158 ($p<0.001$). There is a stronger correlation between body mass and systolic blood pressure readings (Figure 4.18), than BMI and systolic blood pressure readings. Body mass is thus a stronger indicator of systolic hypertension than BMI.

12. Diastolic blood pressure

The diastolic blood pressure of the subjects ranged from 40mmHg to 110mmHg (Figure 4.19). The mean for the subjects' diastolic blood pressure was 71.23mmHg (SD = 10.43mmHg). The mean diastolic blood pressure for the girls was 70.22mmHg (SD = 10.55mmHg). The mean for the boys' diastolic blood pressure was 72.11mmHg (SD = 10.27mmHg).

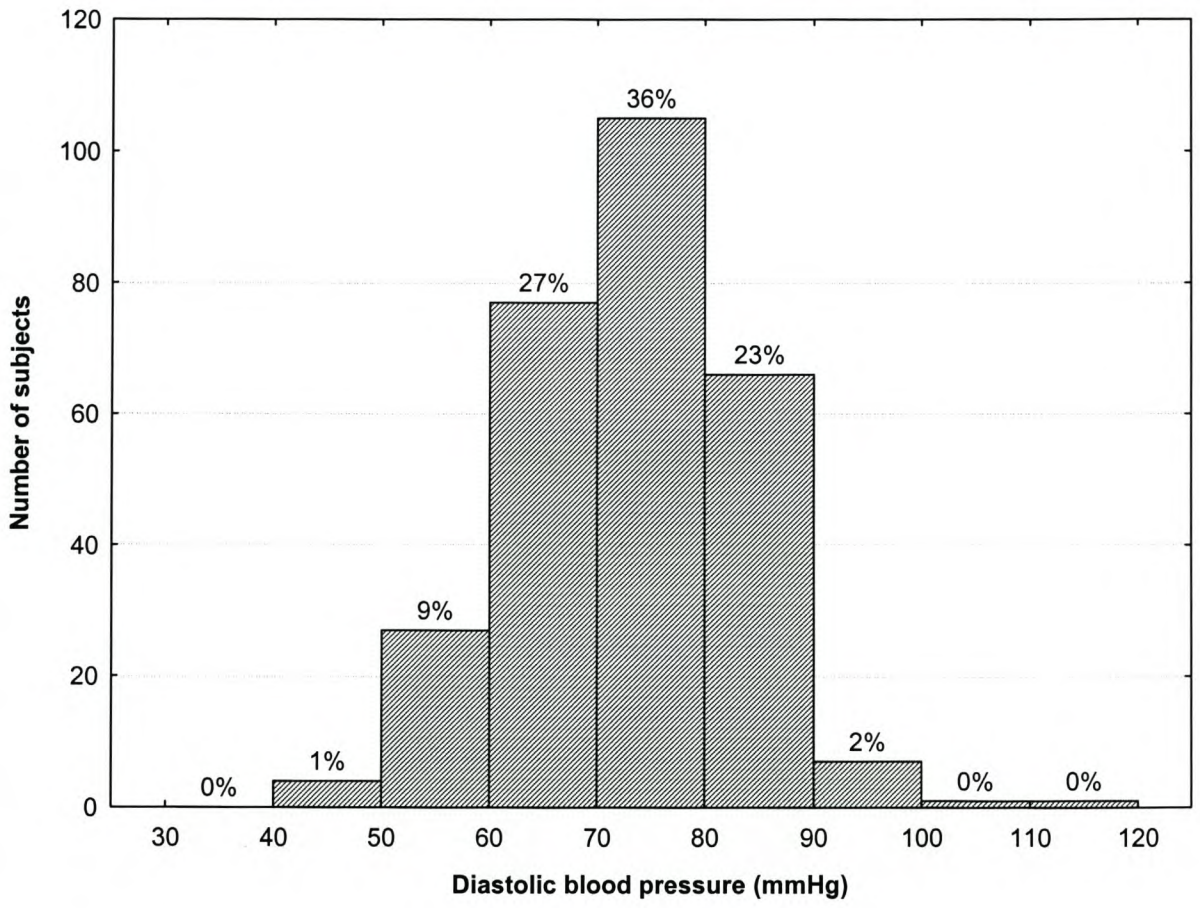

Figure 4.19 A frequency histogram of diastolic blood pressure.

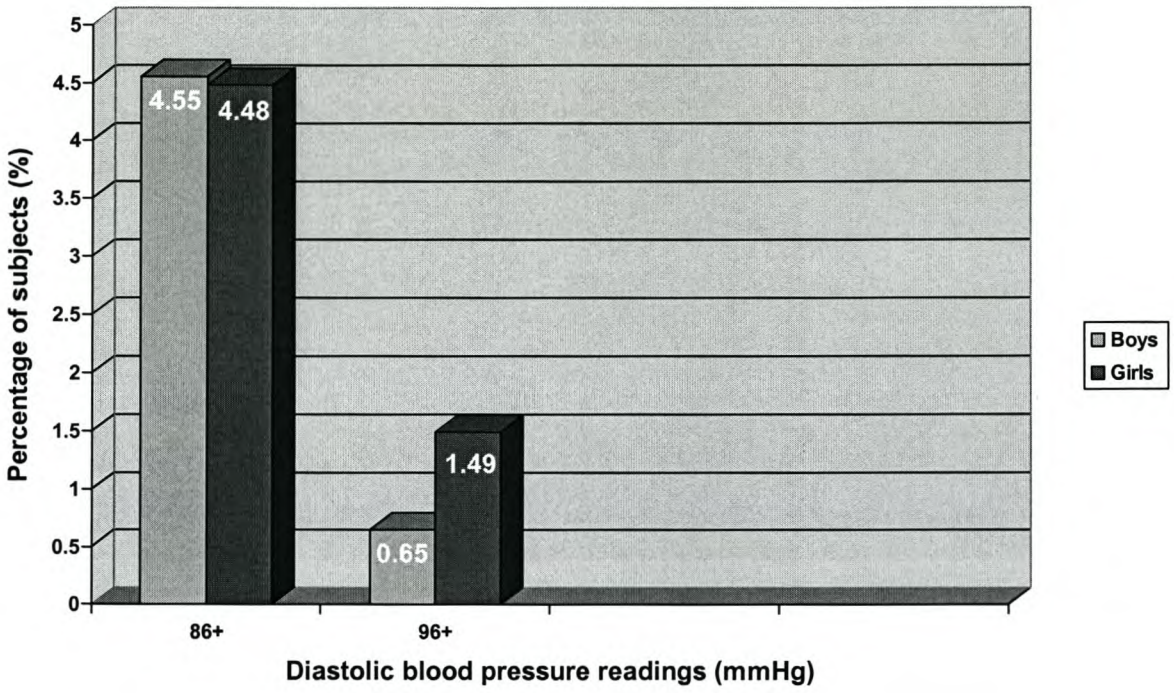

Figure 4.20 A graphic illustration of the percentage of subjects with moderate and severe diastolic hypertension.

In figure 4.20 the number of boys and girls with moderate and severe diastolic hypertension, respectively, is illustrated. In other words, 4.51% of all the subjects tested had a diastolic blood pressure reading indicating mild to moderate hypertension. Of all the subjects tested, 1.04% had a diastolic blood pressure reading indicating severe hypertension. Two of the boys and two of the girls with diastolic hypertension did not have systolic hypertension.

When observing the systolic as well as the diastolic hypertension, there were 56 (19.44%) children with mild to moderate hypertension and 16 (5.56%) children with severe hypertension.

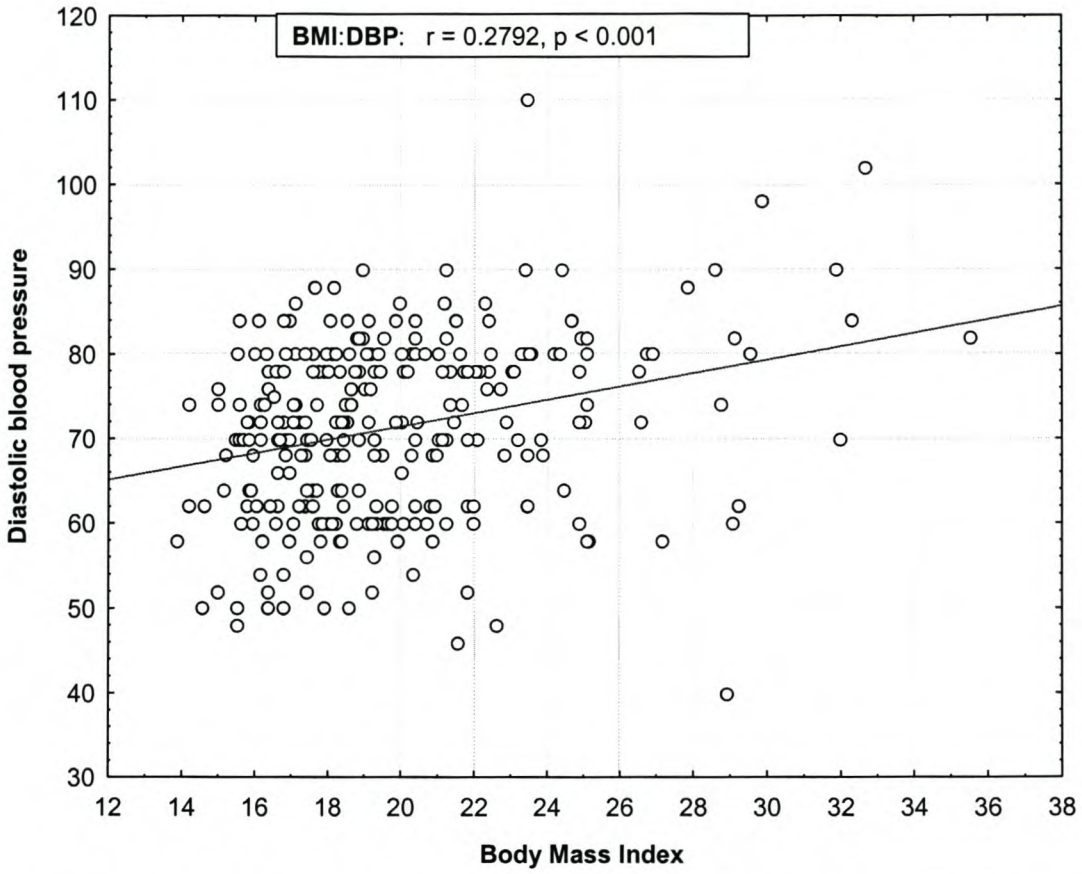

Figure 4.21 A scatter plot to illustrate the correlation between diastolic blood pressure and body mass index.

The correlation between diastolic blood pressure readings and BMI (Figure 4.21) was 0.2792 ($p<0.001$), not as strong as the correlation between BMI and systolic blood pressure readings.

13. $\dot{V}O_2max$

The $\dot{V}O_2max$ of the subjects ranged from 20.61ml.kg.min^{-1} to 76.05ml.kg.min^{-1} (Figure 4.22). The mean for the subjects' $\dot{V}O_2max$ was 48.34ml.kg.min^{-1} (SD ± 11.90ml.kg.min^{-1}). The mean $\dot{V}O_2max$ for the girls was 38.25ml.kg.min^{-1} (SD ± 4.84ml.kg.min^{-1}). The mean for the boys' $\dot{V}O_2max$ was 57.05ml.kg.min^{-1} (SD ± 8.92ml.kg.min^{-1}). The girls' $\dot{V}O_2max$ ranged from 23.7ml.kg.min^{-1} to 49.19ml.kg.min^{-1} and the boys' $\dot{V}O_2max$ ranged from 20.61ml.kg.min^{-1} to 76.05ml.kg.min^{-1}.

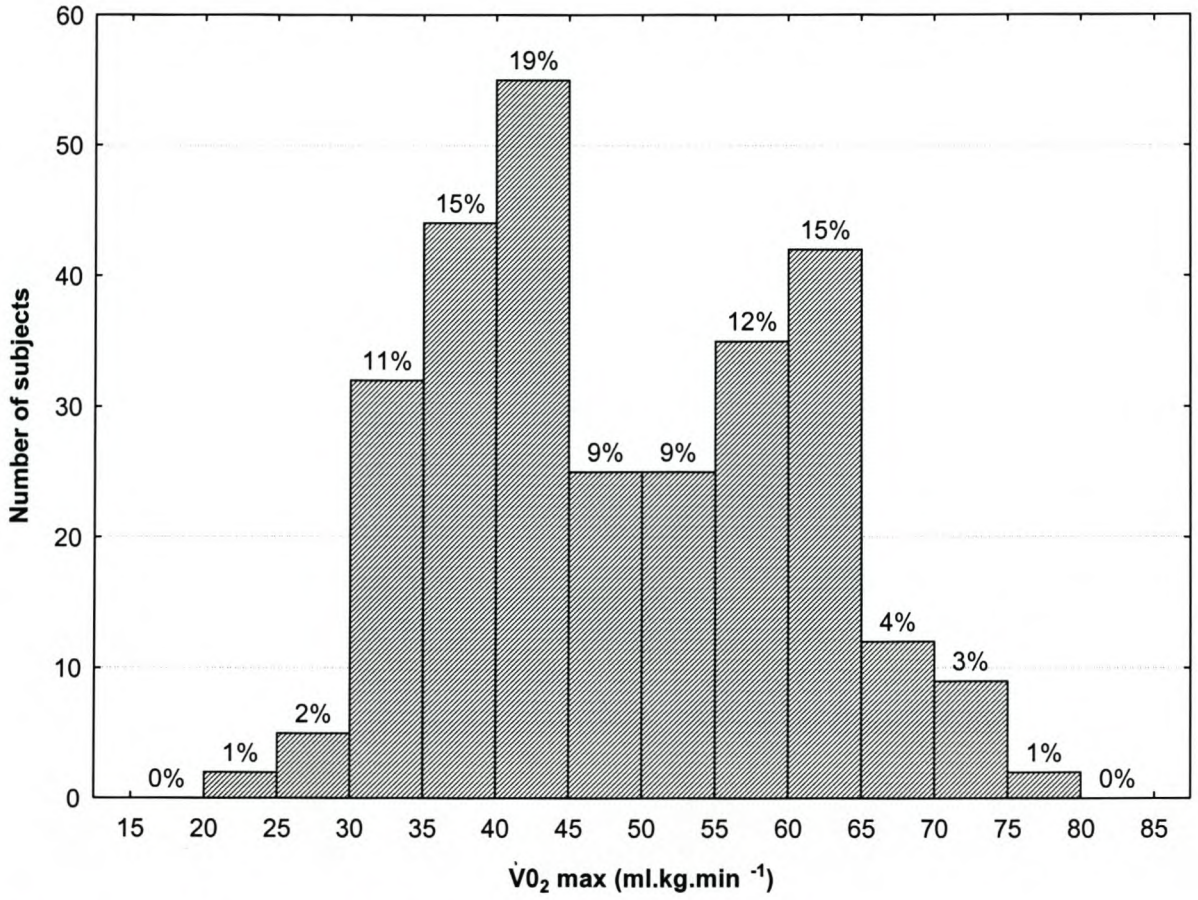

Figure 4.22 A frequency histogram of the subjects' $\dot{V}O_2$ max.

From the frequency histogram (Figure 4.18) there are clearly two modes in the subjects $\dot{V}O_2$ max results. When looking at the means of the subjects there is clearly a big difference between the mean $\dot{V}O_2$ max of the boys and girls, which explains the two modes. This is confirmed by the fact that there was a statistically significant difference between the $\dot{V}O_2$ max of the girls and the $\dot{V}O_2$ max of the boys ($p<0.001$).

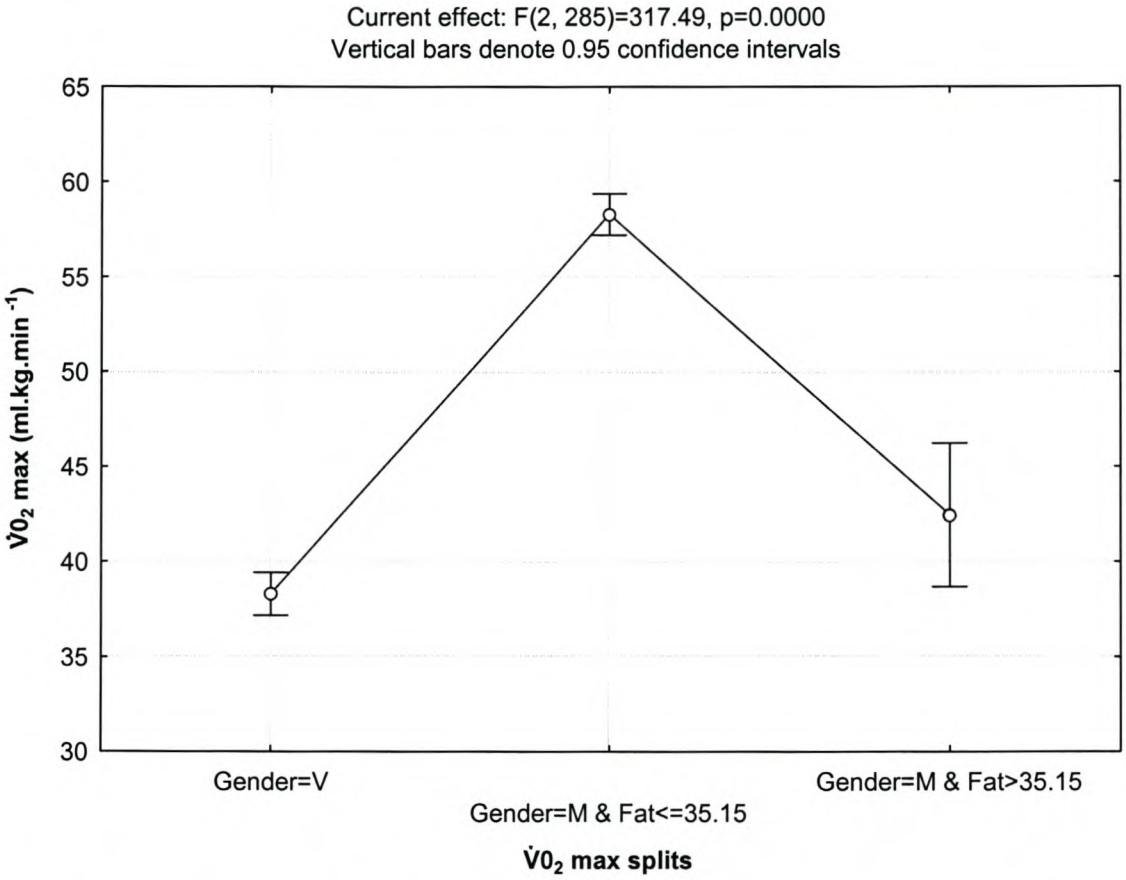

Figure 4.23 ANOVA graph illustrating $\dot{V}O_2$max splits.

In figure 4.23 a regression tree analysis was done to identify homogenises groups of children with regards to $\dot{V}O_2$ max (Appendix F). This analysis was done for the group as a whole. The mean $\dot{V}O_2$ max of the girls was 38 ml.kg.min^{-1}. This was significantly lower than the $\dot{V}O_2$ max of the boys with a percentage body fat lower than 35.15% ($p<0.001$). There was no significantly difference between the $\dot{V}O_2$ max of the girls and the VO_2 max of the boys with a percentage body fat higher than 35.15% ($p=0.12$). For the boys, percentage body fat had an influence on $\dot{V}O_2$ max. $\dot{V}O_2$ max is statistically significantly lower in boys with a percentage body fat higher than 35.15% than that of boys with a percentage body fat lower than 35.15% ($p<0.001$).

14. Heart rate

The heart rate (HR) of the subjects ranged from 84 beats per minute to 228 beats per minute after completion of the three-minute step test (Figure 4.24). The mean for the subjects' HR was 138.58 beats per minute (SD ± 25.85 beats per minute). The mean HR for the girls was 149.33 beats per minute (SD ± 26.54 beats per minute). The mean for the boys' HR was 129.23 beats per minute (SD ± 21.26 beats per minute). The girls' HR ranged from 90 beats per minute to 228 beats per minute and the boys' HR ranged from 84 to 216 beats per minute.

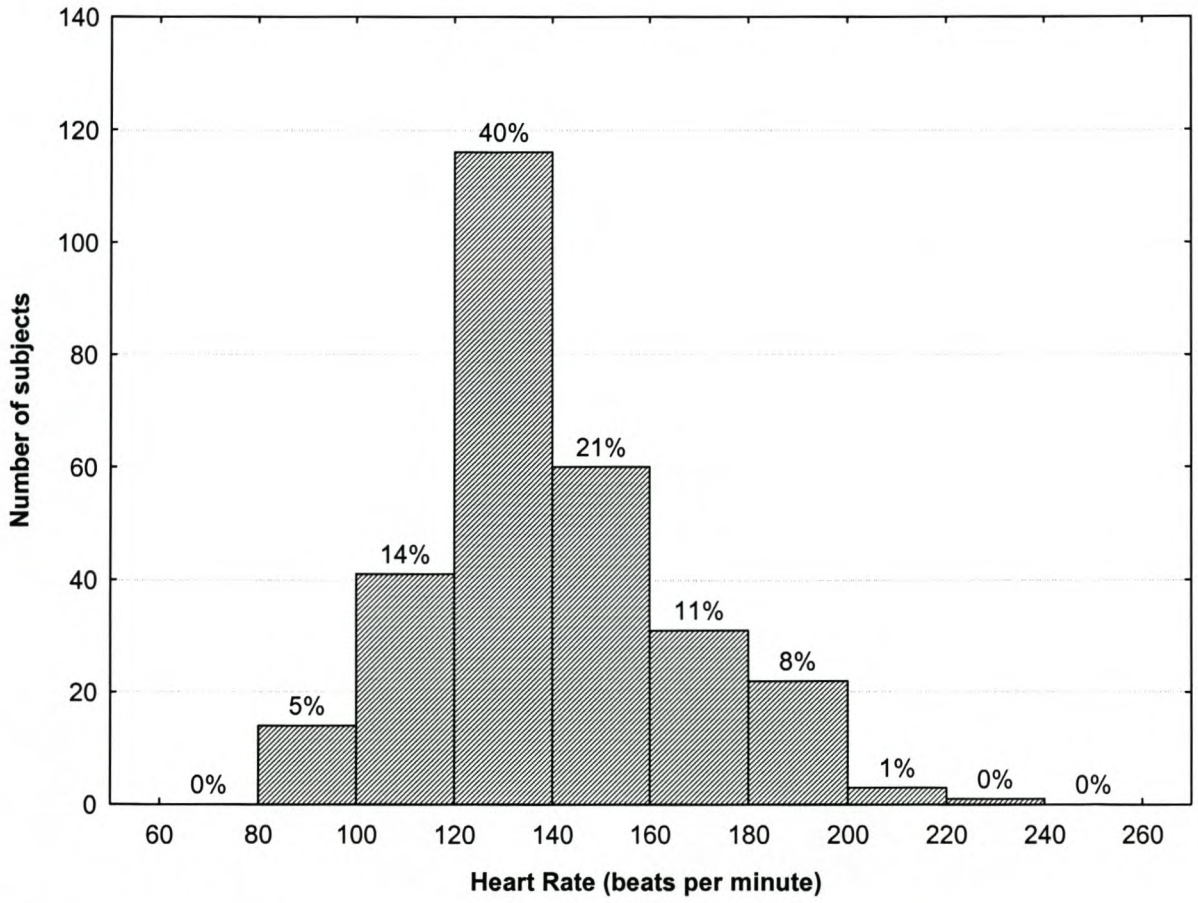

Figure 4.24 A frequency histogram for subjects' heart rate after step test.

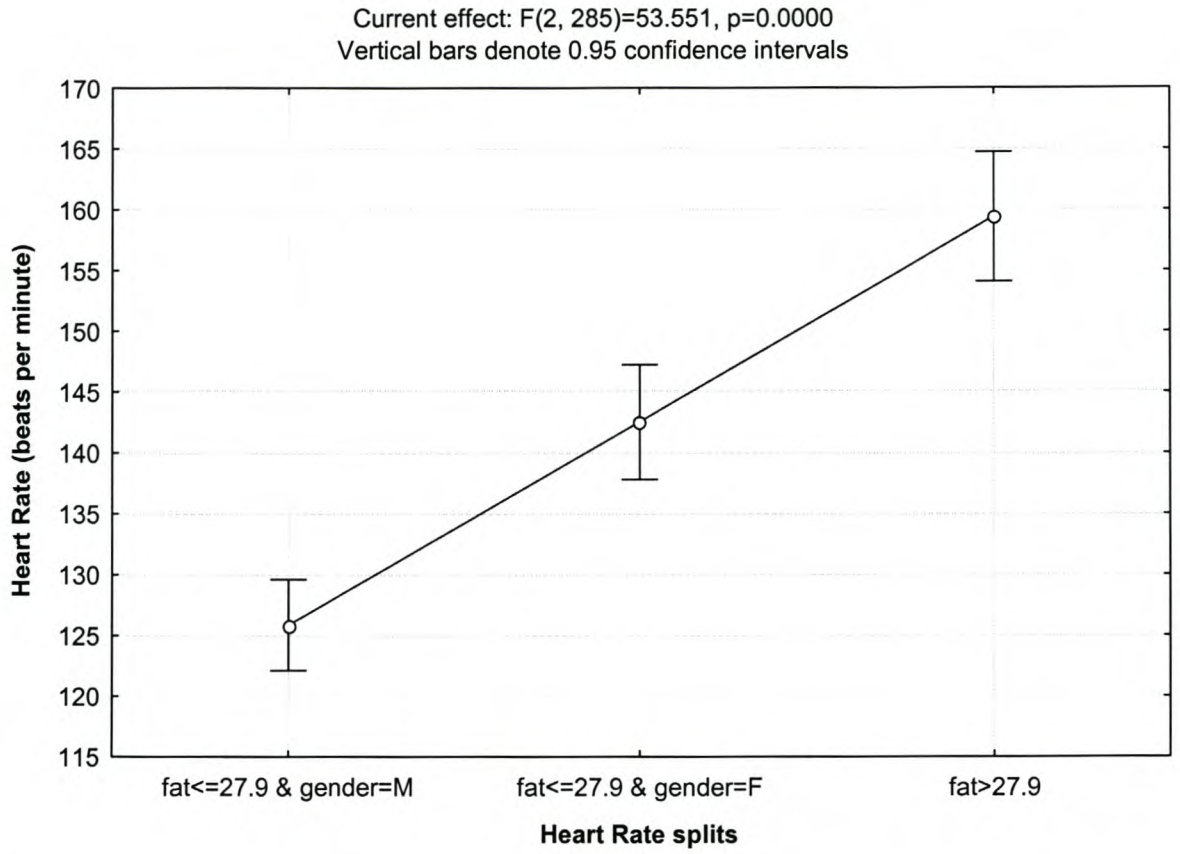

Figure 4.25 ANOVA graph illustrating heart rate splits.

As in figure 4.15 and 4.23, regression tree analysis was also used in figure 4.25 to identify homogenises groups, with regards to heart rate. Percentage body fat and gender were identified as the determents of heart rate (Figure 4.25).

In subjects with a percentage body fat lower or equal to 27.9%, there was a statistic significant difference between the mean heart rate of the boys and the mean heart rate of the girls ($p<0.001$). The mean heart rate of the boys, with a percentage body fat lower than 27.9%, was 126 beat per minute. The mean heart rate of the girls with a percentage body fat lower than 27.9% was 146 beats per minute. In subjects with a percentage body fat higher than 27.9%, there was no distinction between the heart rate of the boys and the heart rate of the girls. The mean heart rate of this group was 159

beats per minute, which was statistic significantly higher than the mean heart rate of the other two groups ($p<0.001$).

Children with a percentage body fat higher than 27.9% thus tend to have higher heart rate readings on a three-minute step test. Higher heart rate readings are an indication of lower physical fitness levels. There is thus a decrease in physical fitness as percentage body fat rises above 27.9%.

The reason for this can be that children with excess body fat are not as physically active as their leaner counterparts, because of being teased or awkwardness of movement. The higher a person's percentage body fat, the more energy is required for movement. It can thus be that some of these children are relatively active, but higher heart rates are possibly because of higher energy requirements.

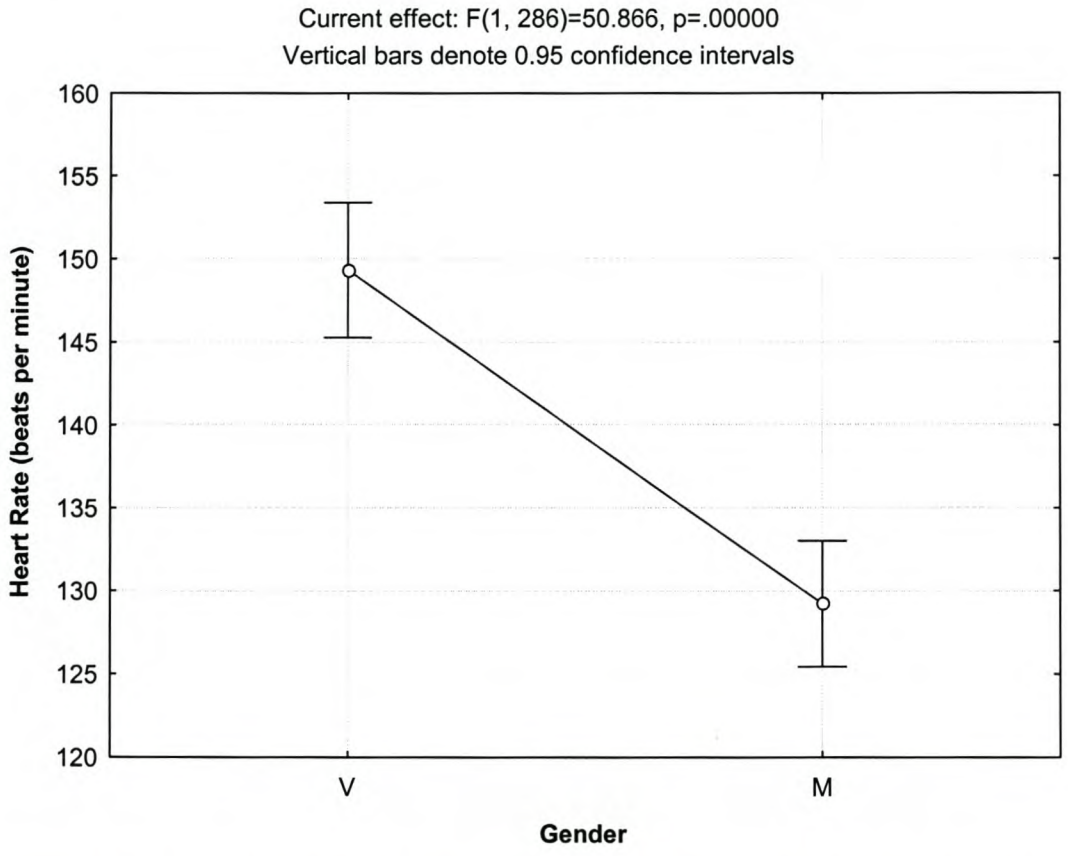

Figure 4.26 ANOVA graph to illustrate gender differences in the heart rate after the three-minute step test.

To ensure that the difference in $\dot{V}O_2$ max (Figure 4.26) between the girls and the boys was not because of the different equations used for the different genders but because of a genuine significant difference between the HR data and thus the physical fitness levels, the difference between the HR data of the boys and girls was calculated. As seen in figure 4.26 there was a significant difference in the HR of the boys and the girls ($p<0.01$). Thus, the different equations used for $\dot{V}O_2$ max reflect the HR data, and thus physical fitness, accurately. The assumption can thus be made that on the average the boys had higher fitness levels than the girls. The fitness test was adapted for the girls (a slower stepping cadence), thus similar HR data could have been expected. Unfortunately this was not the case, and thus rating the girls higher on this risk factor.

15. Family history

As seen in figure 4.27, out of the 288 subjects tested, 221 (77%) had a family history associated with an increased risk for the development of CAD. Only 67 (23%) of the subjects' family history do not increase their risk for the development of CAD. Out of the 221 subjects with a family history that increase their risk for the development of CAD, 105 (47.51%) were girls and 116 (52.49%) were boys. There is no statistic significant correlation between gender and a family history associated with an increased risk for the development of CAD amongst the subjects tested ($p>0.05$).

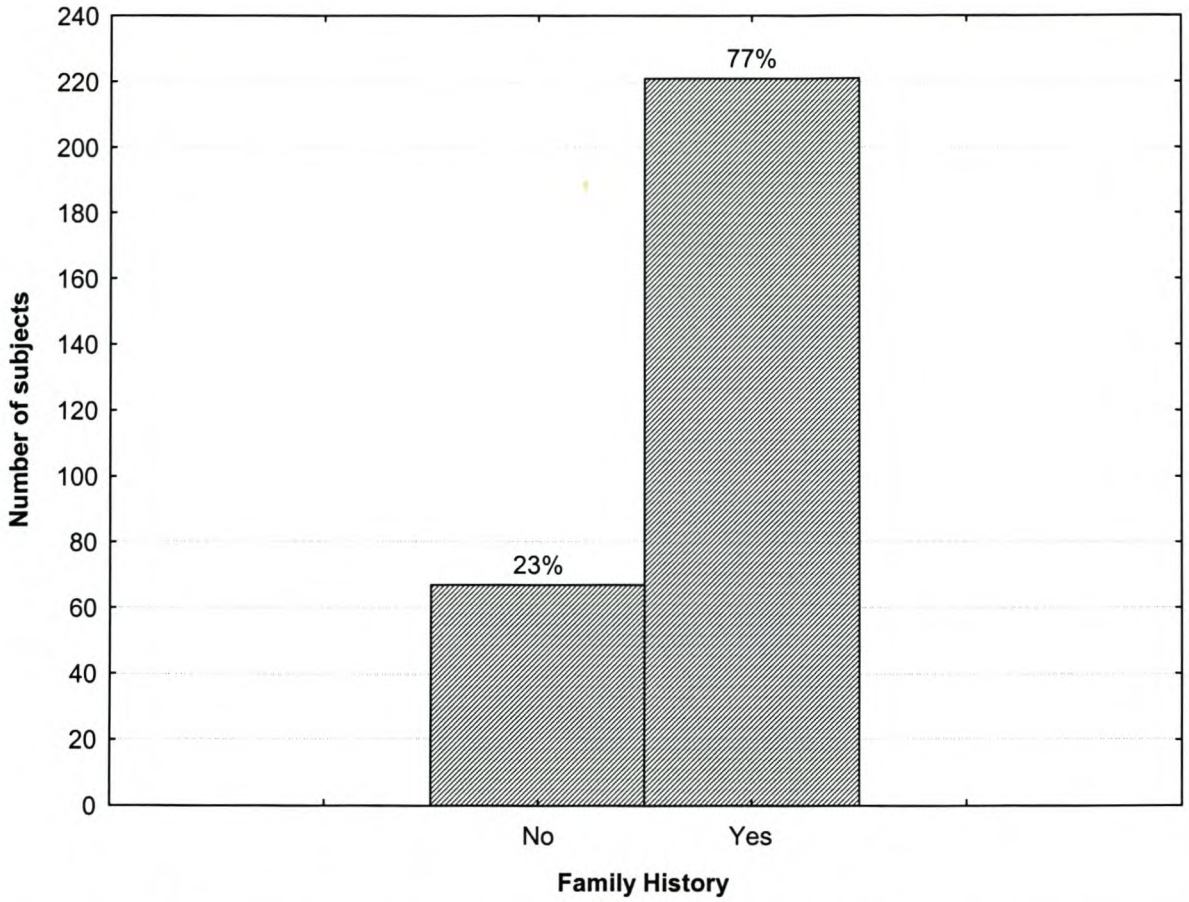

Figure 4.27 The percentage subjects with a family history associated with an increased risk for the development of CAD.

Family history is a primary CAD risk factor. A very high percentage of subjects had a family history associated with an increased risk for CAD. A family history that increases the risk for CAD includes the following:

A primary family member (parents, grandparents) that had a heart attack or stroke before the age of 65 for women and before the age of 55 for men.

A history of sudden death (of natural causes) of any primary family members.

A history of hypertension amongst any primary family members.

A history of high cholesterol amongst any primary family members.

Figures 4.28 to 4.31 gives more detail on the family history of the subjects.

16. Family history of heart attacks and strokes

Out of the 288 subjects tested, 125 (43%) had one or more primary family member that had a heart attack or stroke before the age of 55 for men or 65 for women (Figure 4.28). More than half, 163 (57%), of the subjects had no family history of heart attacks or strokes before these ages.

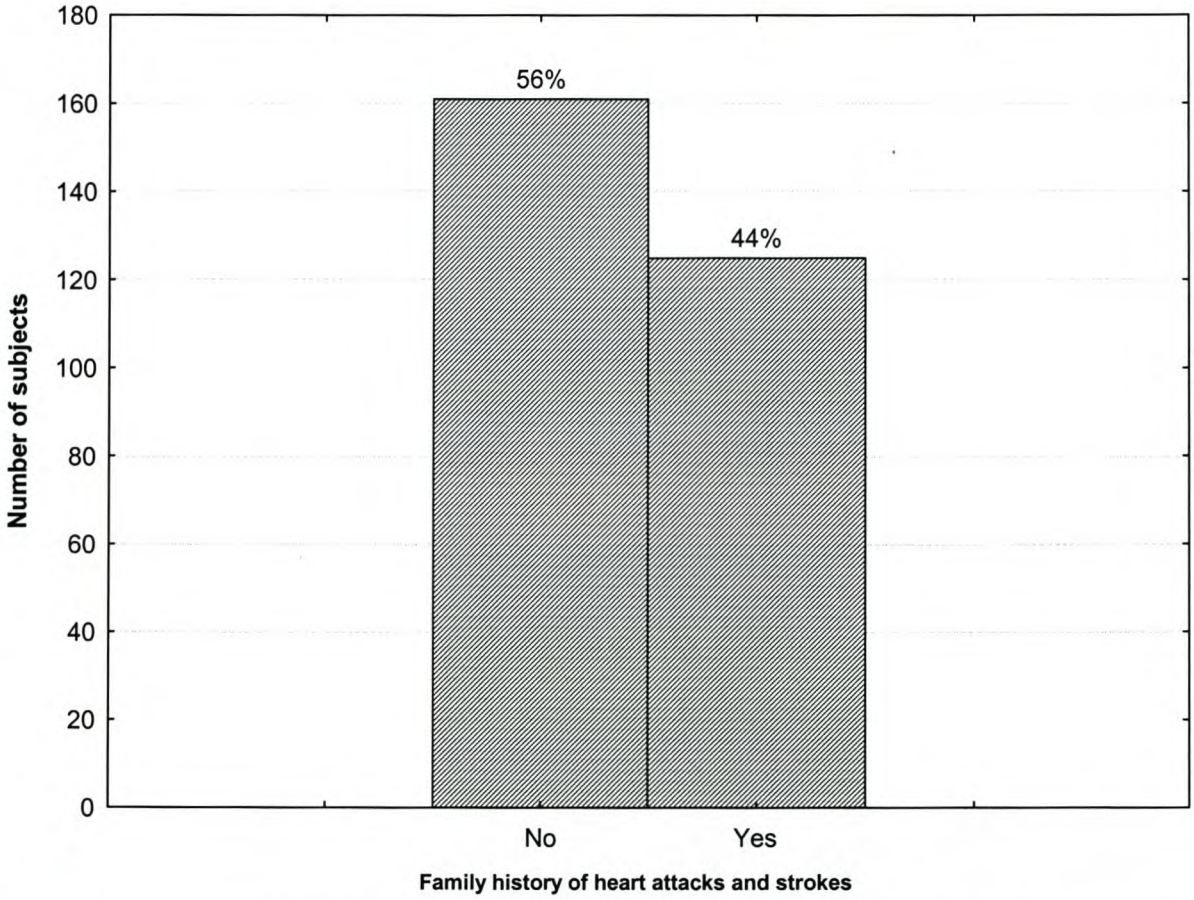

Figure 4.28 Percentage of subjects with one or more primary family member that had a heart attack or stroke before the age of 55 for men or 65 for women, respectively.

A very high percentage of the subjects (47%) had a family history of heart attacks and stokes that increase their risk for heart attacks and stokes. The number of subjects with family members that had a heart attack or stroke is actually much higher, but for the

purpose of this study a history of heart attacks or strokes was only considered a CAD risk when and if it occurred before the age of 55 for men and before the age of 65 for women.

17. Family history of sudden death

Out of the 288 subjects tested, 49 (17%) reported sudden deaths, of natural causes, in their families (Figure 4.29). Of all the subjects, 239 (83%) had no sudden deaths in the family.

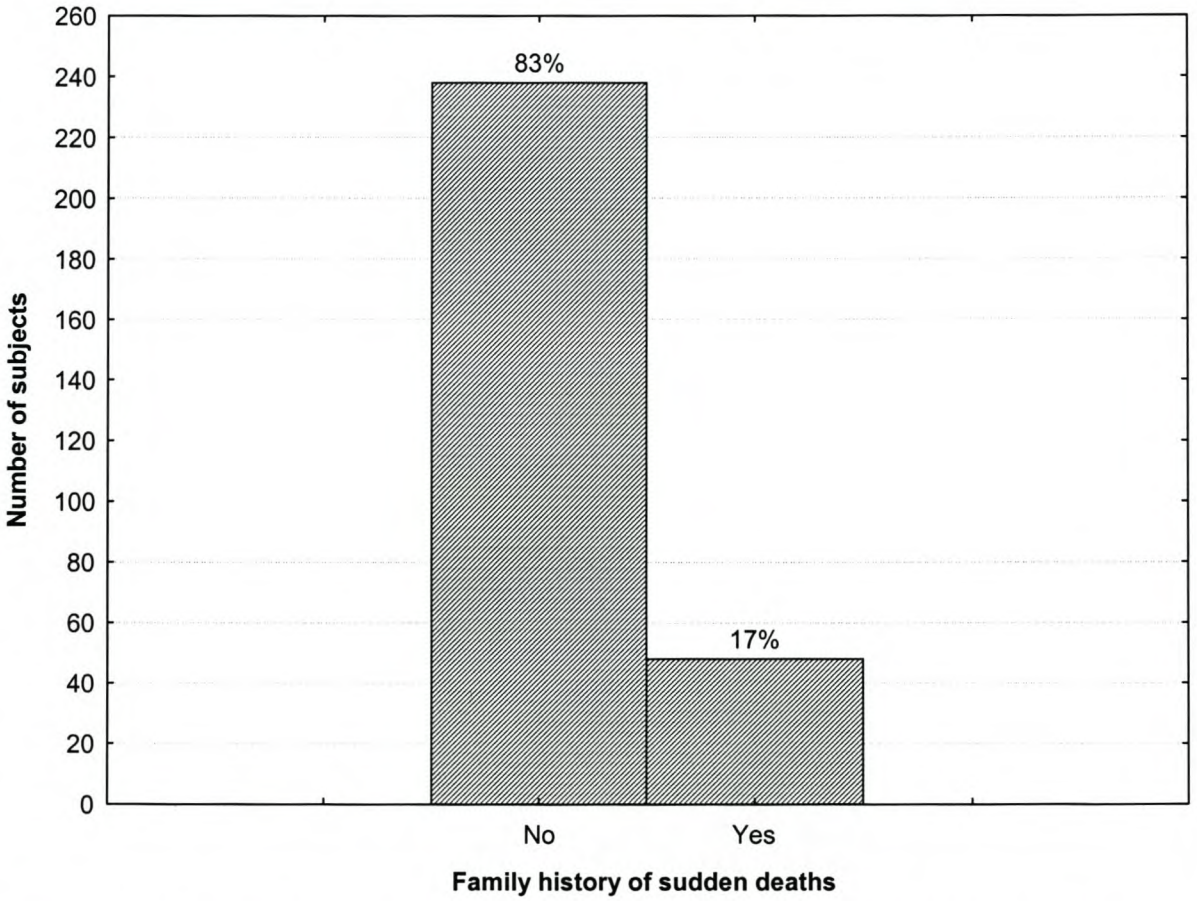

Figure 4.29 The percentage subjects where there was a sudden death, of natural causes, in the family.

Most natural sudden deaths are CAD related. The exact cause of death of a family member is not always known, hence why it was included in the questionnaire.

18. Family history of hypertension

Out of the 288 subjects tested, 170 (59%) had a family history of hypertension (Figure 4.30). Only 118 (41%) of the subjects had no hypertension in their families.

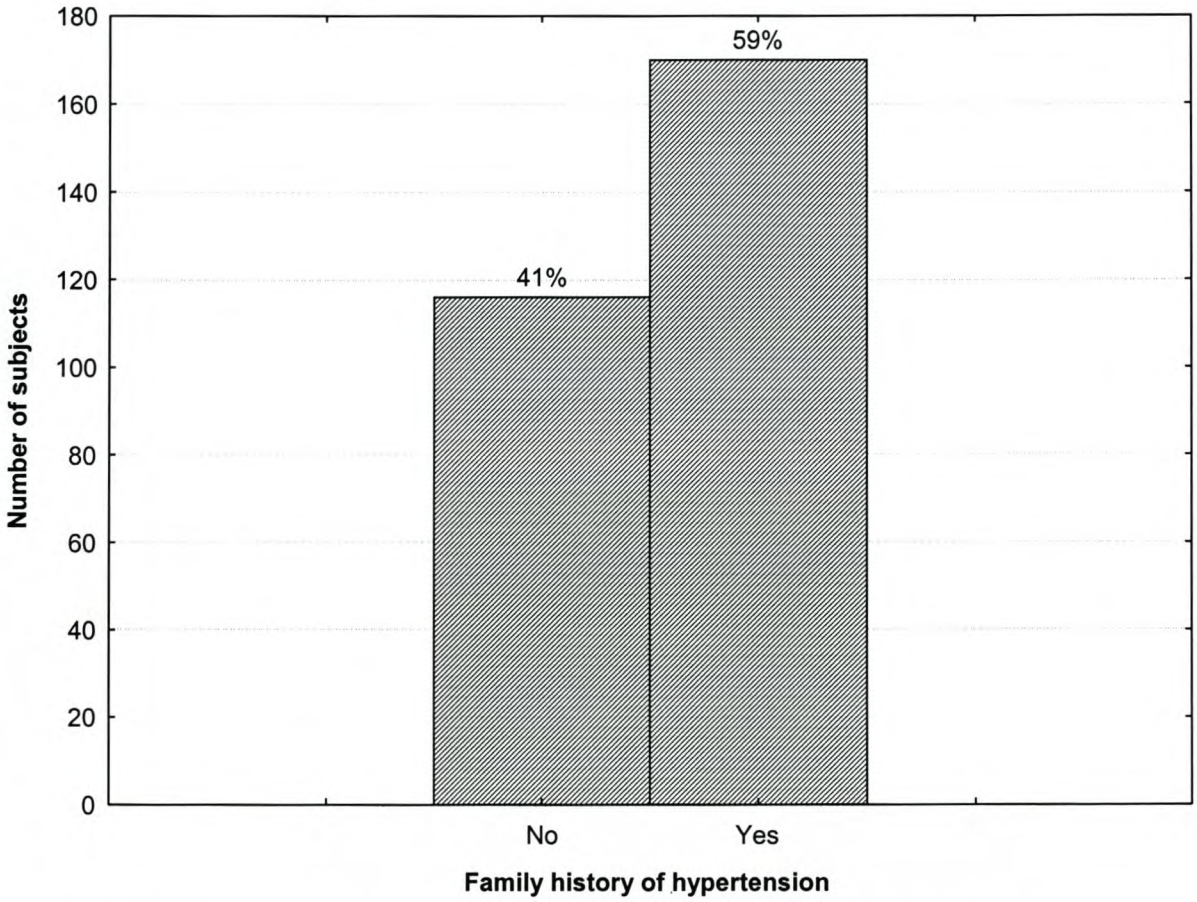

Figure 4.30 The percentage subjects with hypertension in their families.

A child with a primary family member with hypertension has a very high change of developing hypertension. The high percentage of subjects with a primary family member with hypertension is an indication that one can expect an increase in the

number of subjects with hypertension in years to come, unless intervention occurs. It is also possible that there are more family members with undiagnosed hypertension.

19. Family history of high cholesterol

Out of the 288 subjects tested, 23 (8%) had a family history of high cholesterol (Figure 4.31). Most of the subjects, 265 (92%), had no family history of high cholesterol.

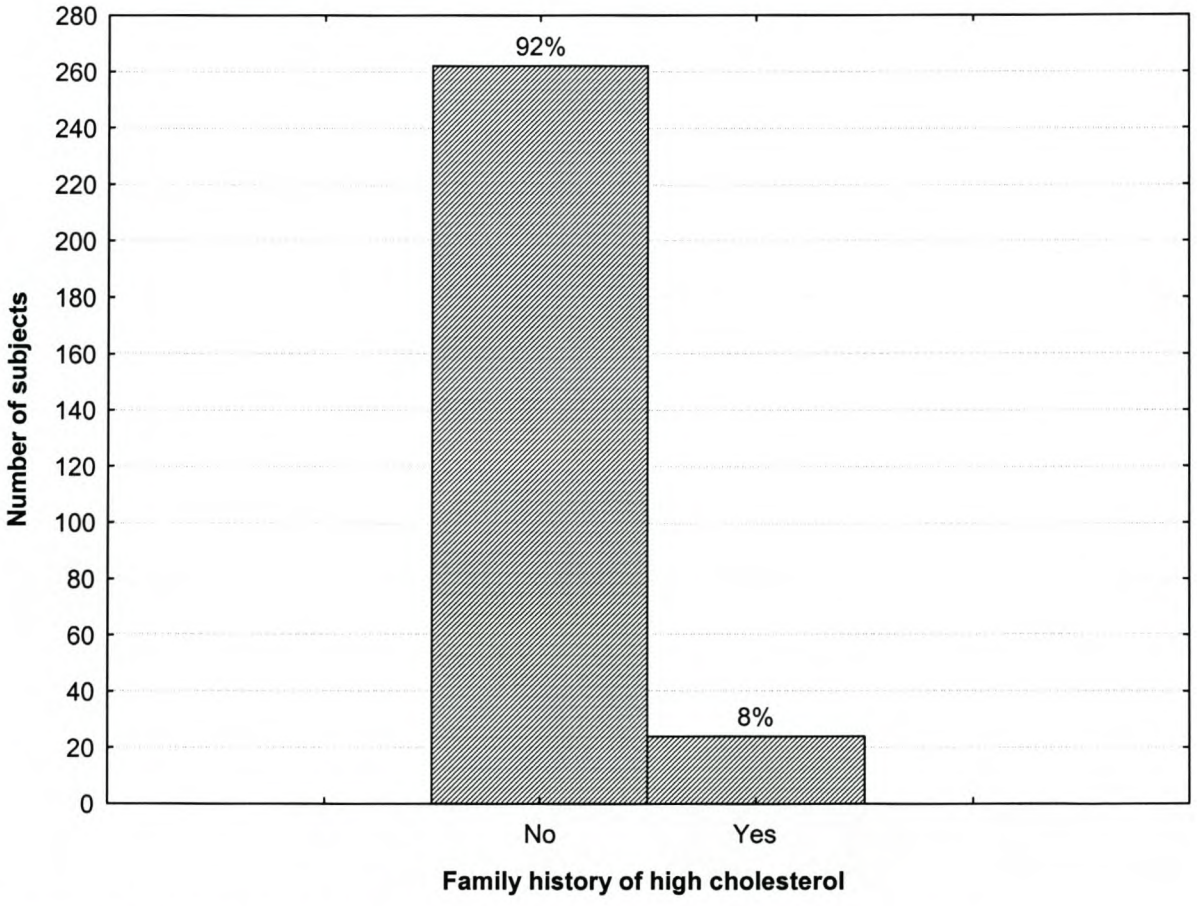

Figure 4.31 Percentage subjects with a family history of high cholesterol.

The 92% that has no family history of high cholesterol also includes a very high percentage of people that do not know their cholesterol levels. Very few of the parents indicated that they have had their cholesterol levels checked.

20. Smoking

According to the children and their parents, none of the subjects smoked. This was determined by the questionnaires completed by the parents and the children. The social stigma connected to smoking as well as the smoking laws, by which it is illegal to sell cigarettes to children under the age of 16, could be the reason why none of the children admitted to ever using tobacco. When looking at the statistics available for the prevalence of smoking amongst South African children, it is most likely that some of the subjects tested actually do smoke.

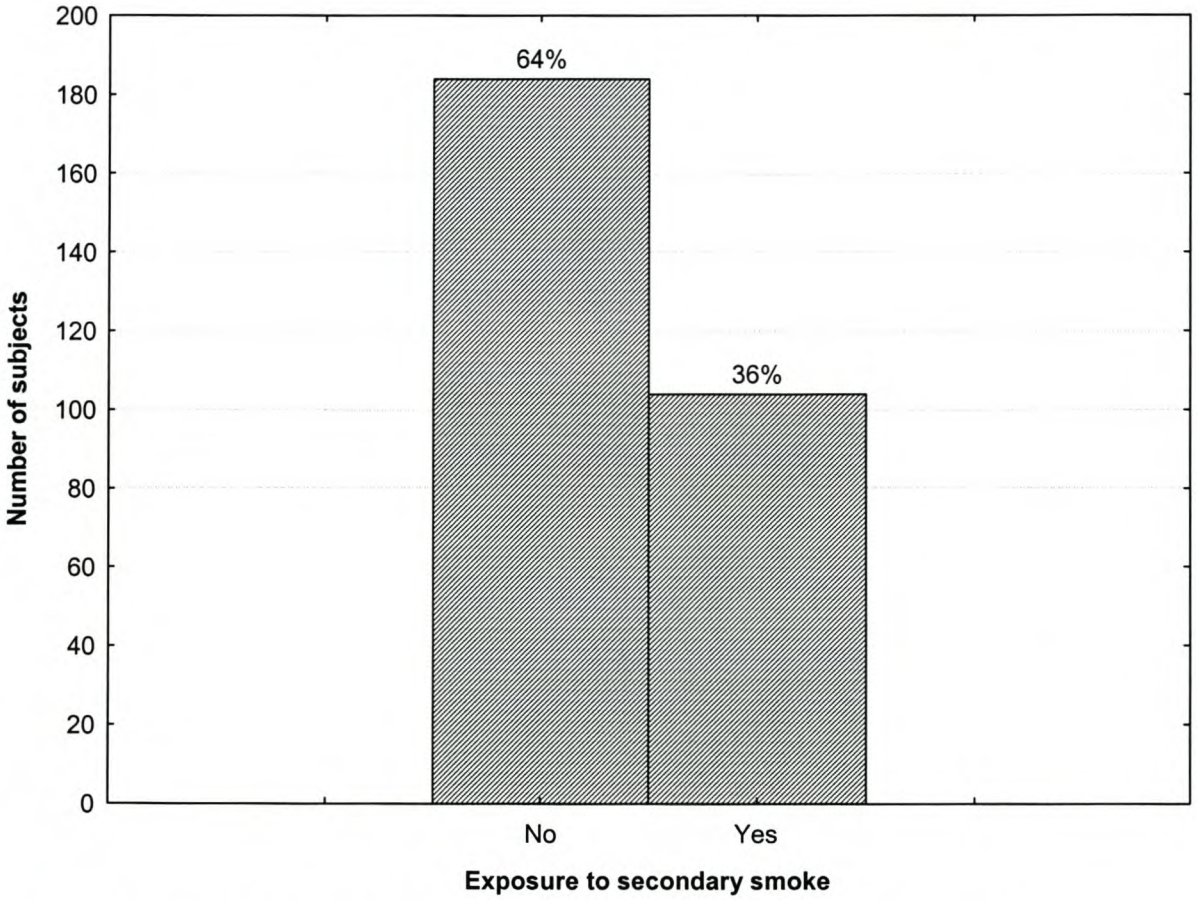

Figure 4.32 The percentage subjects that are exposed to secondary smoke.

Out of the 288 subjects tested, 104 (36%) were exposed to secondary smoke on a daily basis (Figure 4.32). More than half, 184 (64%), of the subjects' parents or caretakers do

not smoke. Out of the 104 subjects that were exposed to secondary smoke, 55 (52.88%) were girls and 49 (47.12%) were boys. In a study done by Steyn and associates (2000), 45.4% of the white subjects in their study were exposed to secondary smoke on a daily basis.

Secondary smoke is also considered a CAD risk factor (Plowman and Smith, 1997:193; Steyn *et al.*, 2000). Outside their home and school environment, most children are exposed to secondary smoke on a daily basis. Adding to this, many children get more frequent and intense exposure in their own homes. This exposure to tobacco smoke increases their risk for CAD in the same way that a smoker's risk is increased. Few parents and caretakers realise this and therefore often smoke in the company of children.

Although it was not determined by the questionnaire, the number of cigarettes smoked in the presence of children will also have an effect on how much their CAD risk had increased. For the purpose of this study, secondary smoke was considered a CAD risk factor if a child was exposed to secondary smoke on a daily basis, due to the smoking habits of his/her parents or caretakers. The daily quantity of cigarettes was not determined.

21. Diabetes mellitus

Out of the 288 subjects tested, only 2 (1%) of the subjects - one girl and one boy - had diabetes mellitus (Figure 4.33). Most of the subjects (286 or 99%) have never experienced any problems with their blood sugar.

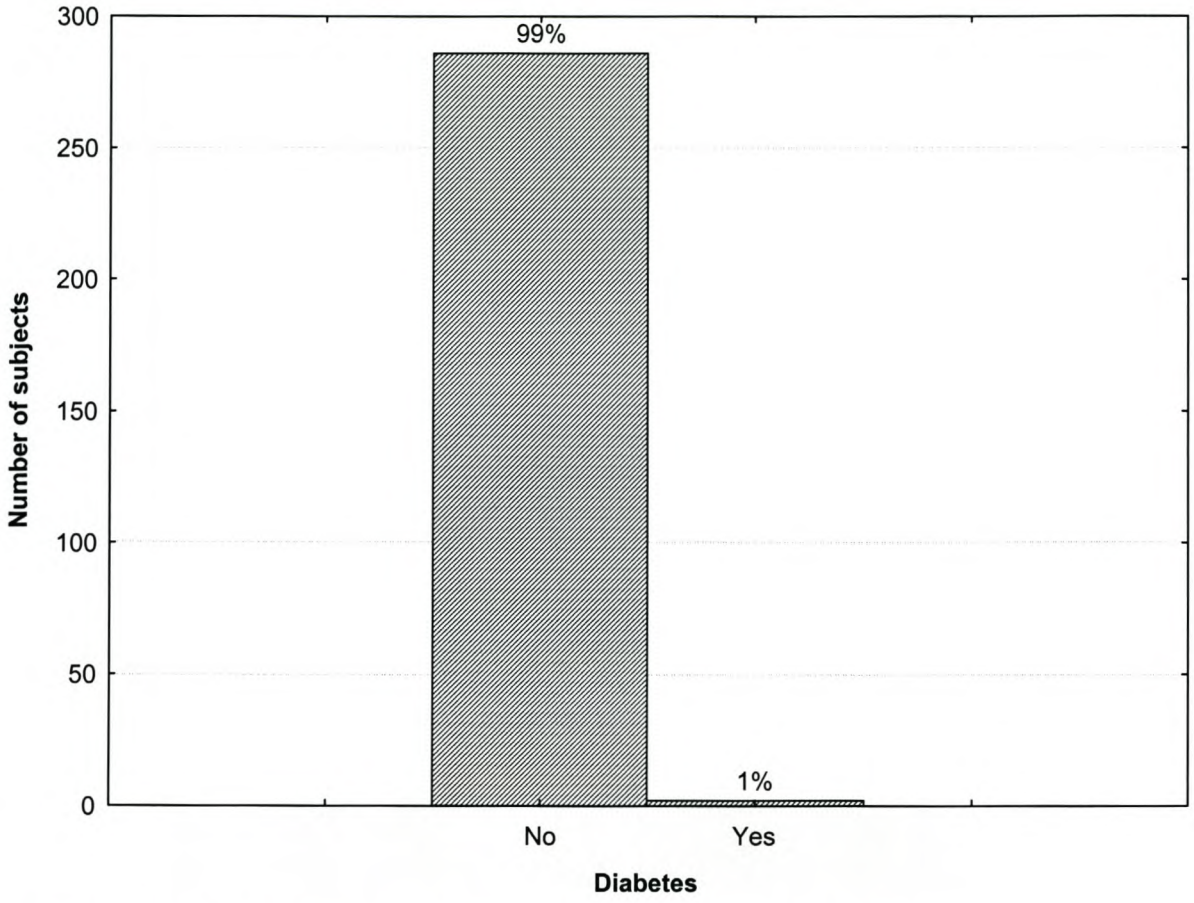

Figure 4.33 The percentage subjects known with diabetes mellitus.

Blood glucose could not be tested because of financial limitations, hence diabetes was assessed though the questionnaire (Appendix C). Only two of the children were taking insulin treatment. Whether this is an accurate reflection of the subject's diabetes status is not sure. The assumption is made that more of the children suffer from some form of undiagnosed blood glucose dysfunction.

22. Family history of diabetes mellitus

Out of the 288 subjects tested, 81 (28%) had a family history of diabetes mellitus (Figure 4.34). Most of the subjects, 207 (72%), had no family history of diabetes mellitus. Out of the 81 subjects with a family history of diabetes mellitus, 38 (46.91%) were girls and 43 (53.01%) were boys.

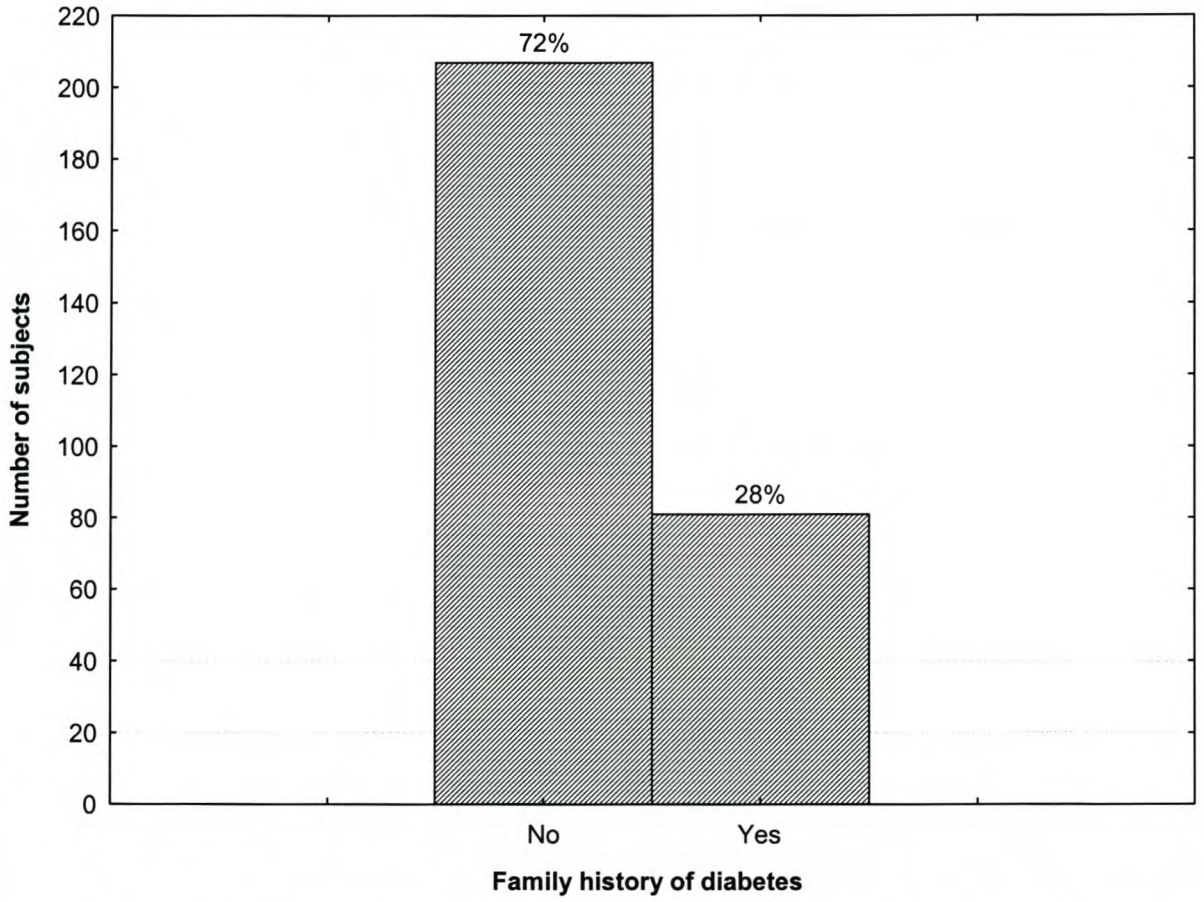

Figure 4.34 The percentage subjects with a one or more primary family members with diabetes mellitus.

Diabetes mellitus (especially Type I) has a very strong heredity component. When looking at the number of subjects with a family history of diabetes (Figure 4.33), it gives more reason to believe that more subjects actually do have diabetes or are at risk to develop it at a later stage in life.

23. Diet

The dietary scores of the subjects ranged from 19 to 55 (Figure 4.35). The mean for the subjects' dietary scores was 34.78 (SD = 6.75). The dietary scores for girls ranged from 19 to 50; the mean for their dietary scores was 33.19 (SD = 6.39). The mean for the boys' dietary scores was 36.17 (SD = 6.43); their scores ranged from 23 to 55.

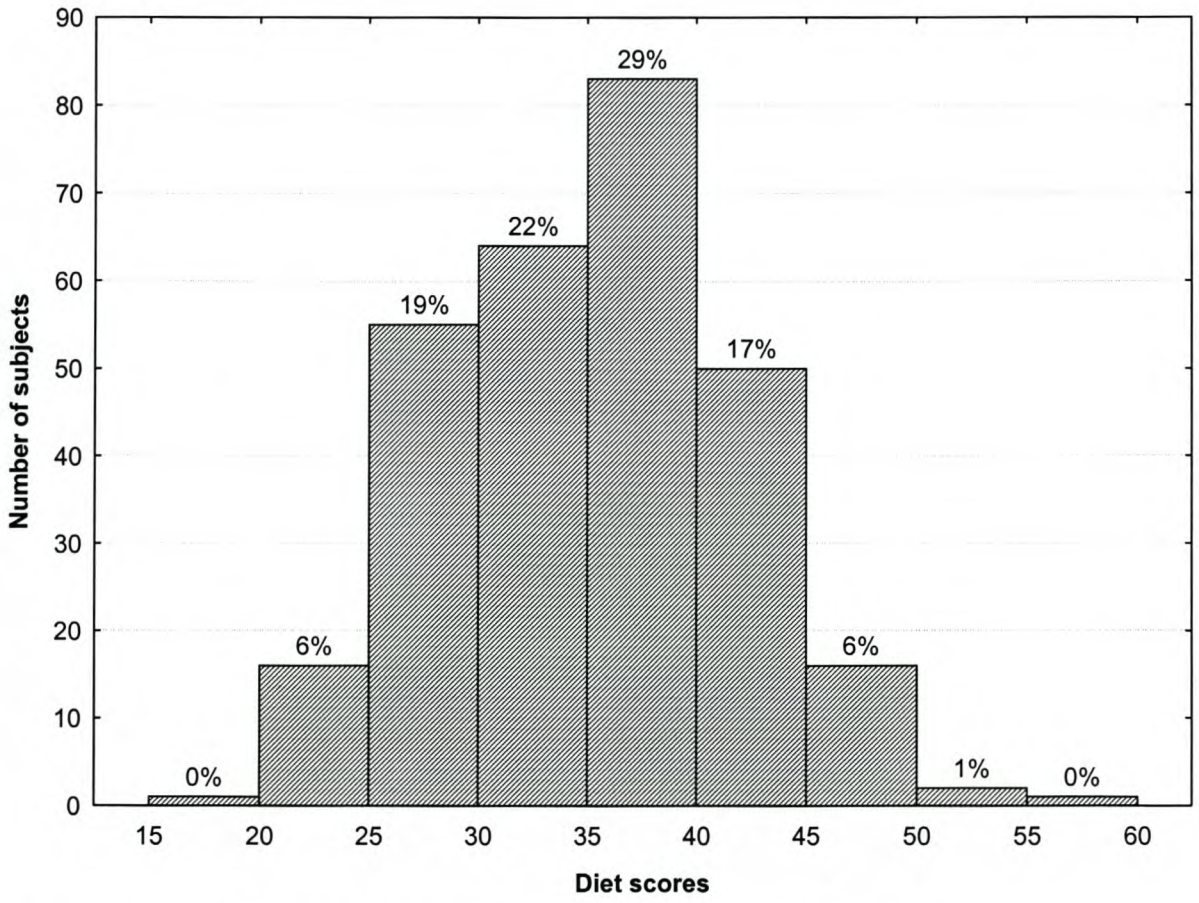

Figure 4.35 A frequency histogram of the subjects' dietary scores.

Subjects with a dietary score of 35 or higher were considered having too high an intake of fat, salt and/or sugar. Fifty three percent of the subjects had a dietary score that indicated a diet that increases their risk for CAD. Unfortunately, the fast paced lifestyle that is adapted from the rest of the world has a negative effect on healthy diets. The

consumption of takeaways and microwave “TV-dinners” is on the increase (Gleick, 1999).

CHAPTER V CONCLUSION AND RECOMMENDATIONS

There is strong evidence in the literature that suggests that childhood coronary risk factors are related to the rate of CAD amongst adults. The prevalence of coronary risk factors amongst children can thus be used as a predictor of the CAD rates for future populations. When looking at the data of this study in this light, it suggests that the prevalence of CAD in South Africa will remain high or even increase.

Most of the world is recognising the problem of CAD and allocating resources to the prevention of this decease. However, the main focus of prevention programmes to date has been the adult populations. The value of childhood prevention programmes should not be disregarded. The sooner children are exposed to lifestyle modifications, the better the likelihood that it will have a positive impact for the duration of their life. Lifestyle modifications for the prevention of CAD include an active lifestyle and a healthy diet. This, however, is not a remedy that should be practised for a short duration of time; it should be a way of life. That is the reason why these behaviours must be implemented at the youngest age possible.

The data in this study suggests that there is a need for national studies amongst children of all age and race groups, repeated at regular intervals, to give a reliable picture of the coronary risk factor status of South African children. Changes in risk profiles should also be monitored over time.

The percentage subjects and the number of risk factors present, in this study, are presented in figure 5.1.

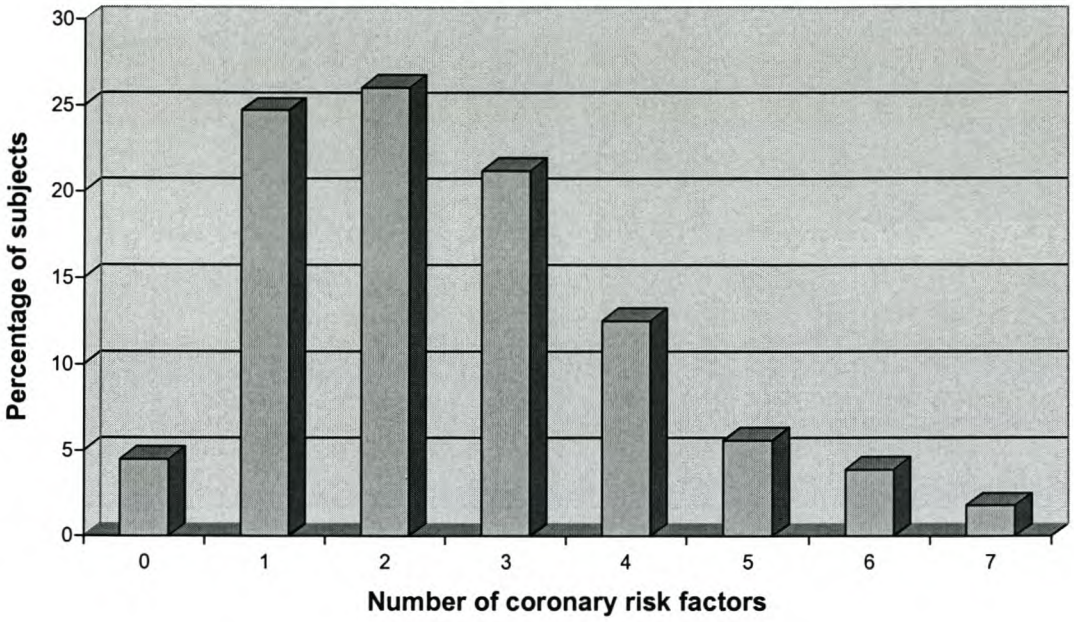

Figure 5.1 A graphic illustration of the percentage of subjects that had a certain number of risk factors.

A brief summary of certain results of this study is presented in figure 5.2.

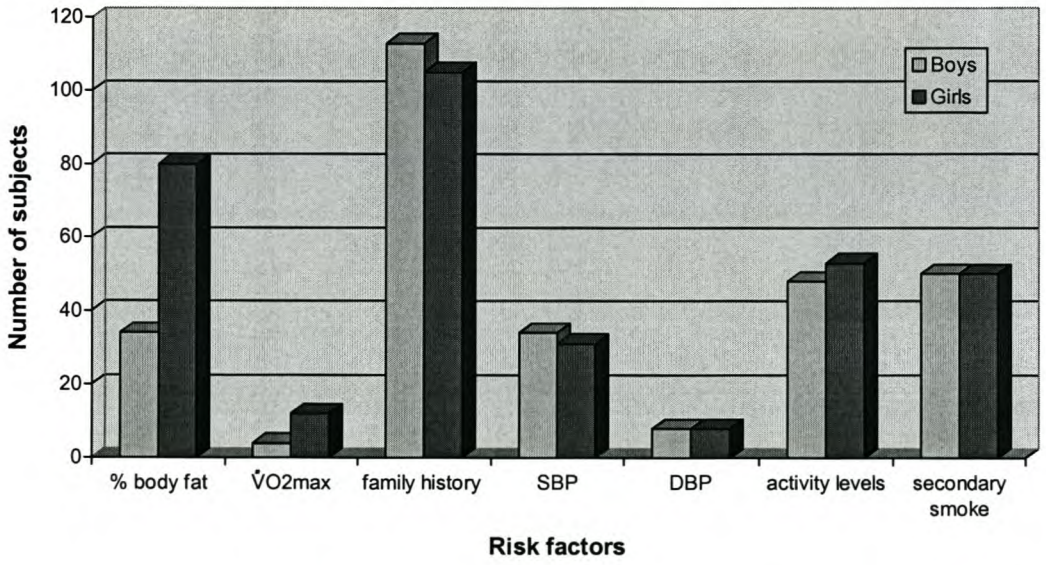

Figure 5.2 The prevalence of certain risk factors amongst subjects in this study.

"% Body fat" refers to the number of subjects with a percentage body fat higher than 20%; "$\dot{V}O_2max$" refers to the number of subjects with a $\dot{V}O_2max$ lower than 31 $ml.kg.min^{-1}$ (Gilliam *et al.*, 1979); "Family history" refers to the number of subjects that has a family history that is associated with an increased risk for CAD; "SBP" refers to the number of subjects that had a systolic blood pressure reading higher than 130mmHg; "DBP" refers to the number of subjects that had a diastolic blood pressure reading higher than 86mmHg; "activity levels" refers to the number of subjects with activity scores lower than eight; "secondary smoke" refers to the number of subjects that are exposed to secondary smoke on a daily base due to smoking habits of parents of caretakers.

Areas of concern are percentage body fat, family history, blood pressure readings, activity levels and exposure to secondary smoke. Although family history cannot be addressed, it is important that people are aware of the risk involved. The new smoking legislation is addressing the issue of secondary smoke exposure. Obesity, activity levels and especially, hypertension in children are not getting the attention needed.

Recommendations

There is a need for a structured, cost-effective approach to health promotion and health education. National objectives and detailed strategies to obtain these objectives should be formulated to aid long-term health promotion. These objectives should address dietary habits as well as physical activity patterns for young children.

Short- and long-term goals and objectives need to be formulated and structured to address these issues. Goals and objectives for 2008 may include the following:

- Reduce the prevalence of obesity in children so that less than 15% of the country's children are classified as overweight.
- Increase the adoption of sound dietary practises and regular physical activity in 50% of overweight children.

- Increase the number of children that engage regularly in light to moderate physical activity for at least 30 minutes a day to 50%.
- Increase the number of children that engage in vigorous physical activity that promotes the development of and maintenance of cardiorespiratory fitness three or more days per week for 20 minutes or more per occasion to 75%.
- Decrease the number of children that engage in no leisure-time physical activity to 15%.
- Decrease the number of children that suffers from any form of hypertension to 15%.

Conventional prevention programmes should also be evaluated. Programmes should be modified to adapt to our fast changing environment. Exciting, new ideas should be implemented to motivate people to adapt to a healthier lifestyle.

The school is in a very favourable position to contribute greatly to the education of children regarding healthy lifestyles. A great percentage of children in South Africa are attending school on a regular basis, which gives the perfect opportunity for exposure to health-promoting activities.

Unfortunately, limited funds available at schools are the cause of downscaling of physical education and extra-curricular sport activities. The time allocated to physical education is insufficient to have a positive impact on children's coronary risk profiles. The downscaling of staff and teachers are to a great extend also responsible for the downscaling of extra-curricular sport activities. The increase in technology is also promoting sedentary behaviour in children.

With the diminishing role that schools play in promoting active lifestyles in children, new approaches should be looked at. Rapid social, economic, and technological changes are indicating a need for change in conventional sport activities. New ways should be looked at to stimulate children into being active. Children that do not excel in sport should be exposed to other physical activities. Not all children are able to participate in the sports traditionally presented at South African schools. Alternative physical activities like hiking, rock climbing and other adventure sports could be implemented as an

alternative. Most countries are recognising the important role of alternative physical activities in increasing the physical activity amongst all children. With the many natural resources and accessible scenic areas, South Africa is presented with the unique opportunity to explore the possibilities of outdoor-education and adventure programmes to advocate physical activity in children. The importance of educating children from a young age regarding the benefits of physical activity and a healthy diet are of vital importance.

Because of the huge financial burden CAD can place on a country's economy, the need for cost effective prevention programmes that can be implemented from an early age cannot be stressed enough.

Limitations

The complications involved in taking blood samples prevented the measuring of blood glucose and cholesterol levels. The ideal would have been to study all of the above risk factors to give a more detailed insight into the risk profiles of children. Although this was not possible, the cost-effective and easy administrated methods used gave a good indication of what the current status of coronary risk factors amongst South African children is. The fact that no expensive equipment was required makes this assessment easy for re-evaluation at a later stage. With this, it is also possible for schools to easily assess the risk profiles of their pupils.

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APPENDICES

Appendix A

First letter to headmasters of schools

Department of Sports Science
Stellenbosch University
Stellenbosch
Matieland
7602
June 2001

_____
_____
_____
_____

Dear Sir/Madam

PERMISSION TO CONDUCT A RESEARCH PROJECT

During August 2001 the following research project in some primary schools in the Western Cape, supervised by the Department of Sport Science, Stellenbosch University, are being conducted in your region:

> *The prevalence of coronary risk factors in children ages 11-13, in the Western Cape.*

Every 34 seconds a person dies from coronary heart disease in the United States alone. That means that coronary heart disease is the number one cause of death in the United States. According to the American Heart Association, 40% of all twelve-year-old children display heart disease risk factors (i.e., obesity, hypertension, high cholesterol, and inactivity). The statistics of South Africa is not readily available. If risk factors are

identified at an early age, children can enroll in prevention programs that could possibly reduce mortality rates due to coronary heart disease per se.

To be able to conduct this study, your co-operation is needed.

Participation in this study will entail the following:
A team comprising of biokinetic interns and sport science students from the Stellenbosch University will visit the school on a pre-arranged day during August 2001. This will be conducted at a time that will keep disruption of the normal school routine to a minimal. We will obtain permission from the children's parents/guardian prior to conducting the survey. Approximately 18 children can be tested in an hour.

We hereby ask permission to include your school in this study. If you require more information we will be happy to come and personally speak to you and your personnel to inform you about the contents and procedures of this study.

Please return the enclosed form or contact us in this regard before 29 June 2001.

Thanking you in advance.

Danelle de Klerk
(Biokinetics Intern)
Tel: (021) 808 4735
biokin@maties.sun.ac.za

Prof. J.G. Barnard
(Head of Biokinetics: Stellenbosch University)
Tel: (021) 808 4718
jgb@maties.sun.ac.za

I, ...(full name), the head master of .....(name of school) hereby give permission for my school to be included in the research project discussed above. The school is willing to allocate ..... hours to you for this purpose. Children in all three age groups \ two of the age groups \ one of the age groups (please circle where applicable) can be tested.

Signed

..... Date:.....\.....\2001

The Head Master

Contact details of the person we need to contact for further arrangements:

.....

.....

.....

.....

.....

.....

.....

Appendix B

Letter to headmasters of participating schools

Department of Sports Science
Stellenbosch University
Private Bag x1
Matieland
7602
26 July 2001

Dear Mr. .....

PARTICIPATION IN RESEARCH PROJECT

Many thanks for agreeing to participate in my research project. The following are a few final arrangements before our visit to your school.

Kindly distribute the enclosed forms to all the children involved in the project. These forms are to be signed by the parents/guardians and returned to the school before our visit. If they can be lodged at the school until our visit, it will facilitate matters greatly. It is very important that all the parents of the children involved sign these forms. No child can be tested without the consent of his/her parents.

The visits to your schools are as follows:
Grade 5 Boys.....
Grade 6 Boys.....
Grade 7 Boys.....
Grade 5 Girls.....
Grade 6 Girls.....
Grade 7 Girls.....

The time allocated for testing are dependent on the number of children tested. Unless we hear from you before ..... we will take this as confirmed.

On the day of out visit we will require an empty classroom or any other room for testing, four tables with two chairs each and two gym benches. All children must be dressed in a short-sleeved shirt and shorts on the day of testing. If there are any problems in this regard, please notify us as soon as possible.

Thank you once again for your co-operation. Please contact us if there are any questions or problems.

Yours faithfully,

_____

Danelle de Klerk
(Biokinetics Intern)

Appendix C

Questionnaire and letter of informed consent

Stellenbosch University

Department of Sport Science: Stellenbosch Biokinetics Centre

During August 2001 the following research project in some primary schools in the Western Cape, supervised by the Department of Sport Science, Stellenbosch University are being conducted in your region:

The prevalence of coronary risk factors in children ages 11-13, in the Western Cape.

This study is aimed at identifying children with risk factors that will increase their risk for coronary artery disease in later life. Early identification of coronary risk factors is of utmost importance for children in these age groups, because intervention is still possible at this age. Participation in this study will thus be to the benefit of your child. The results of this study will also have a positive contribution to research in this field. Participation in this study is not compulsory and is entirely up to the parents or guardian of the child.

All information is confidential and will only be used for research purposes.

Where applicable, complete in print or mark with a cross (x).

PERSONAL INFORMATION OF THE CHILD

Name:_____Date:_____/_____/2001

Date of birth: (dd/mm/yy)___/___/___ Gender:

Boy	Girl

The following questions will help to evaluate your child's risk for coronary artery disease. Please answer all questions to the best of your ability.

FAMILY HISTORY

1. Has a parent of the child had a heart attack or stroke? Yes____No____
2. Has a grandparent of the child had a heart attack or stroke before the age of 65? Yes____No____

If either of the questions above were answered with "yes", please answer the following questions:

Who had a heart attack or stroke (child's mother, father, maternal grandmother or grandfather, paternal grandmother or grandfather) and at what age? _____Age:___

3. Is there a history of sudden, unexplained deaths in the family? Yes____No____
4. Does anyone in the family have high blood pressure? Yes____No____
5. Does anyone in the immediate family have diabetes? Yes____No____
6. Do the parents smoke cigarettes? Yes____No____
7. What are the parents' cholesterol levels?

   Mother_____Father_____ Date measured: ____/____/_____

OTHER

8. Does your child participate in organised sport or strenuous exercise more than twice a week?
   Yes____No_____

   If "yes", what kind of sport/exercise does your child do and how many times a week does he/she participates in it?_____

   _____

   _____

9. How many hours a day does your child spend watching television or in front of the computer? _____

10. Does your child smoke? Yes____No_____

11. Has your child had any major illnesses? Specify: _____

    _____

    _____

12. Is your child taking any medicines? Specify: _____

    _____

    _____

13. Has your child had his/her cholesterol levels measured? If "yes", what was it and when was it measured? _____Date:_____/_____/_____

14. How many regular meals does your child eat in a day? _____

DIET

1 = never 2 = some times 3 = most of the time 4 = always

17.	My child always puts extra salt on his/her food	1	2	3	4
18.	My child will eat sweets instead of meals	1	2	3	4
19.	My child prefers fried food above cooked or steamed	1	2	3	4
20.	My child eats high fat margarine on his/her bread	1	2	3	4
21.	My child eats the following in excessive amounts:				
	chips	1	2	3	4
	chocolate	1	2	3	4
	ice cream	1	2	3	4
	more than two spoons of sugar in cereal	1	2	3	4
22.	My child drinks the following in excessive amounts:				
	full cream milk	1	2	3	4
	carbonated drinks	1	2	3	4
	sweetened fruit juices	1	2	3	4
	coffee	1	2	3	4
	tea	1	2	3	4
	2 or more spoons of sugar in coffee/tea	1	2	3	4

I (parent/guardian) hereby give permission for my child _____ (name and surname of child), to take part in the study mentioned above. I assume that the persons involved in the project are well trained and will always act in the best interest of my child. I will not hold the Stellenbosch University or the persons involved responsible for any unforeseen circumstances.

Name of parent/guardian: _____ Date:_____/_____/2001

Signature: _____Witness: _____

If you have any questions, please contact any of the following persons:

Danelle de Klerk (Biokinetics Intern)
Tel: (021) 808 4735
biokin@maties.sun.ac.za

Prof. J.G. Barnard (Head: Biokinetics Centre)
Tel: (021) 808 4718
jgb@maties.sun.ac.za

Appendix D

Data collection form

Stellenbosch University

Department of Sport Science: Stellenbosch Biokinetics Centre

SECTION A

PERSONAL INFORMATION

Name and surname:.....

Test date...../...../2001 Date of birth...../...../.....

Gender:

Boy	Girl

Home language:.....

Age of menache:.....

Name of school:.....

Grade:.....

Contact number(s) for parents: (h).....

(w).....

(cell).....

For office use only:

W	B	C	I	A

Ander:.....

SECTION B: Antropometric measurements

Height (m):.....

Body mass (kg):.....

Skinfold measurements (mm): Tricep:.....

Subscapular:.....

Circumferences (cm): Abdominal:.....

Hip:.....

For office use only:

Sum of skinfolds (mm):.....

Fat percentage (%):.....

BMI:.....

W-H ratio:.....

Very low	
Low	
Optimal range	
Moderately high	
High	
Very high	

SECTION C: Physical activity

1. Do you participate in organised sport or strenuous activity? Yes.....No.....

2. If "yes", name the type of sport or exercise.....

3. What is the duration of each sesssion (circle the applicabe number)?

   1 = ½ hour

   2 = 1 hours

   3 = 2 hours

   4 = > 3 hours

4. How many times a week do you exercise?

   1 = 1

   2 = 2

   3 = 3

   4 = >4

5. How do you get to and from the school and sport activities?

   2 = walk

   2 = cycle

   0 = motor/bus/train/taxi

6. If you walk or cycle to school (as mentioned above), what is the distance?

   0 = do not walk or cycle

   1 = 0-1 km

   2 = 2-3 km

   3 = >3 km

7. How many hours a **day** do you spend infront of the television/computer?

   4 = ½ hours

   3 = 1-2 hours

   2 = 2-3 hours

   1 = >3 hours

SECTION D: Other risk factors

Resting blood pressure:

1..... *For office use only:* .....

2.....

three-minute-step test

HR for 10s (pre):..... $HR.min^{-1}$ (pre):.....

HR for 10s (5-15s of recovery):..... $HR.min^{-1}$ (recovery):.....

For office use only:

HR for 10s (5-15s of recovery):

Low	Bo 26
Somewhat low	25-26
Average	24-23
High	22-21
Very high	Under 21

$\dot{V}O_2max$ ($ml.kg^{-1}.min^{-1}$):.....

Girls:

$\dot{V}O_2max\ (ml.kg^{-1}.min^{-1}) = 65.81 - [0.1847 \times HR.min^{-1}\ (recovery)]$

Boys:

$\dot{V}O_2max\ (ml.kg^{-1}.min^{-1}) = 111.33 - [0.42 \times HR.min^{-1}\ (recovery)]$

Appendix E

Box plots

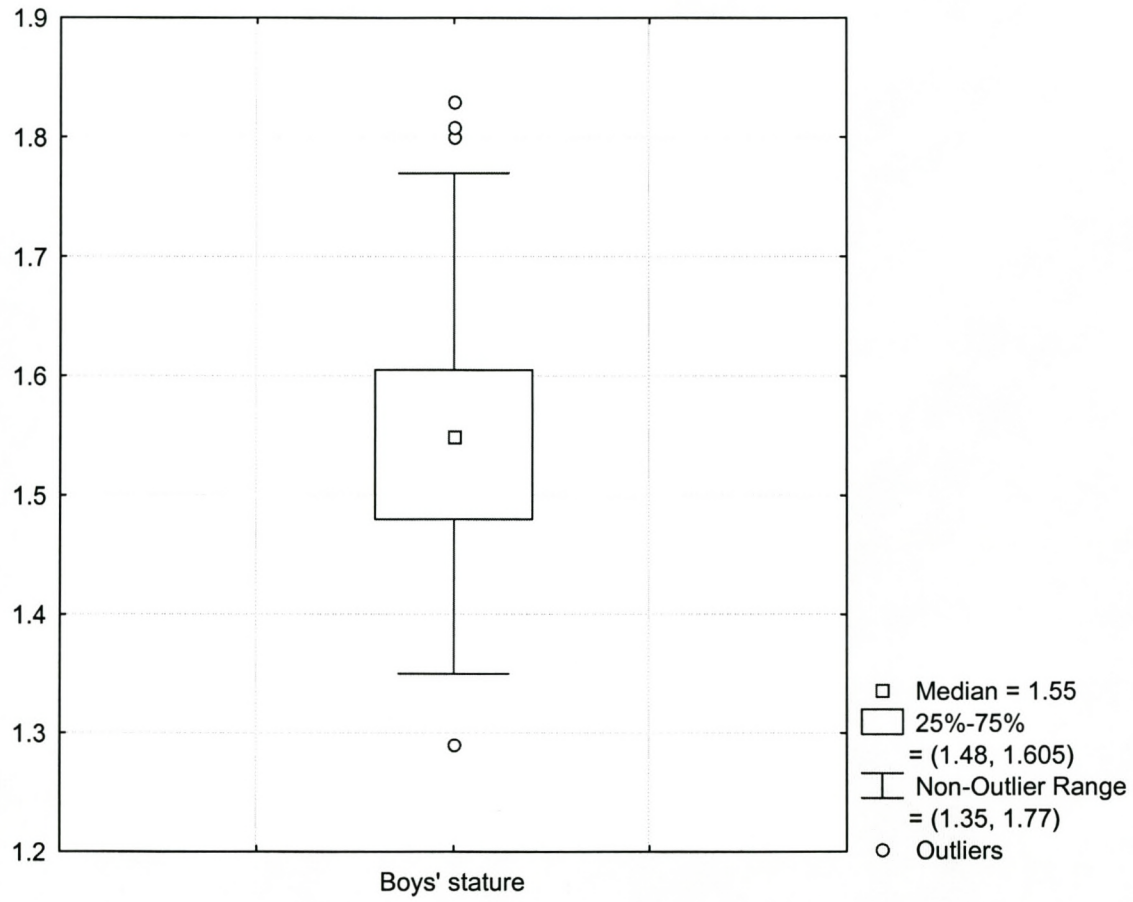

Figure E.1 A box plot illustrating the stature of the boys.

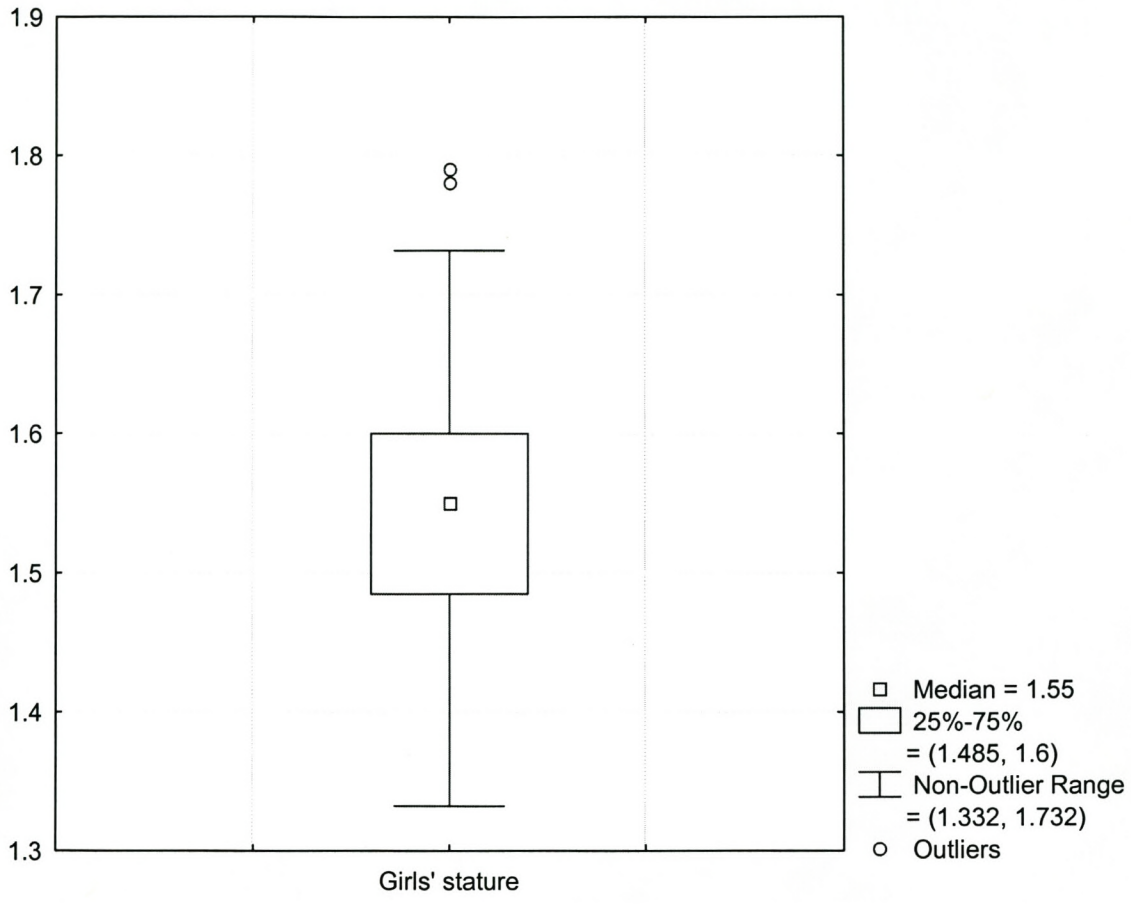

Figure E.2 A box plot illustrating the girls' stature.

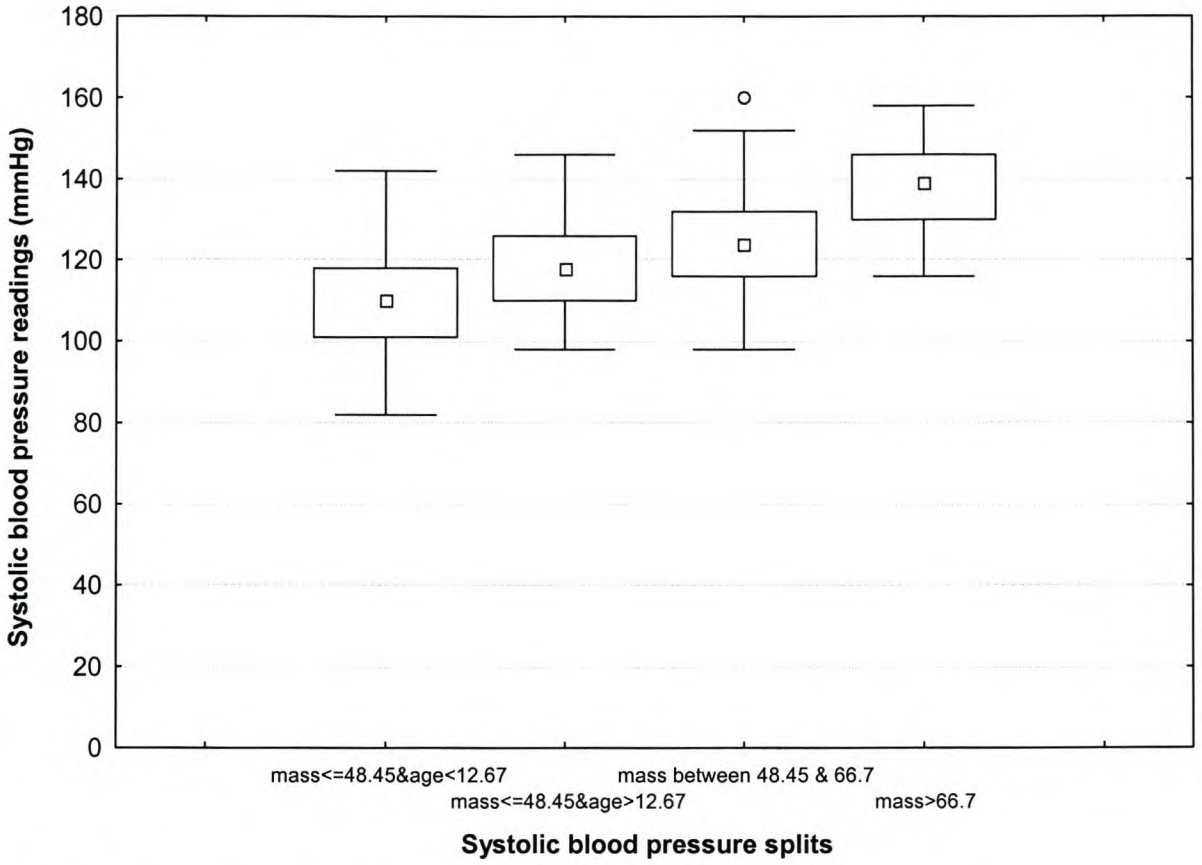

Figure E.3 A box plot illustrating systolic blood pressure splits.

Systolic blood pressure for subjects under the age of 12.67 and with a body mass lower or equal to 48.45kg ranged from 82mmHg to 142mmHg. The median for this group is 116mmHg. The 25th and 75th percentile is 101mmHg and 118mmHg, respectively. Systolic blood pressure for subjects above the age of 12.67 and with a body mass lower or equal to 48.45kg ranged from 98mmHg to 144mmHg. The median for this group is 120mmHg. The 25th and 75th percentile is 110mmHg and 124mmHg, respectively. Systolic blood pressure for subjects with a body mass between 48.45kg and 66.7kg ranged from 82mmHg to 146mmHg. There is one subject with a systolic blood pressure of 160mmHg in this group, it is seen as an outlier. The median for this group is 124mmHg. The 25th and 75th percentile is 116mmHg and 126mmHg, respectively. Systolic blood pressure for subjects with a body mass higher than 66.kg ranged from 116mmHg to 158mmHg. The median for this group is 140mmHg. The 25th and 75th percentile is 110mmHg and 144mmHg, respectively.

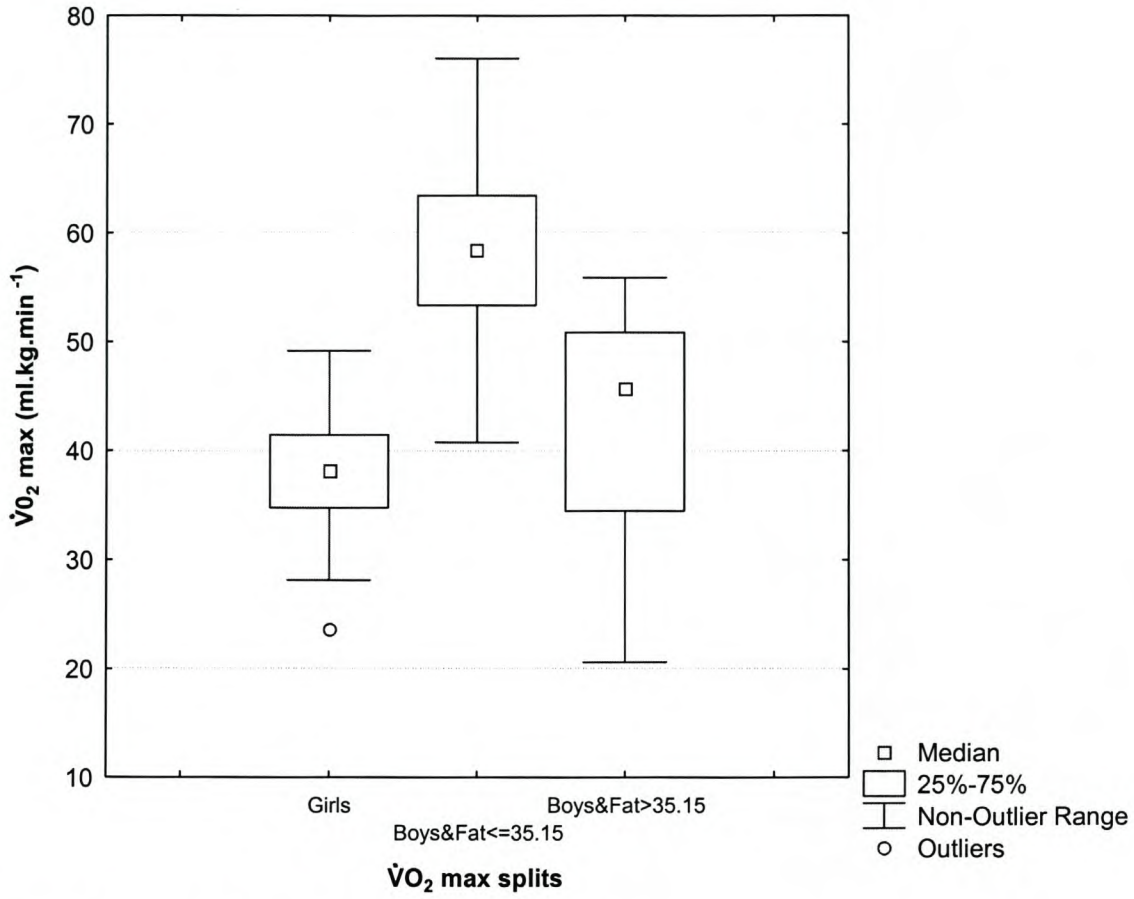

Figure E.4 A box plot illustrating $\dot{V}O_2$ max splits.

According to Figure E.4, the minimum and maximum $\dot{V}O_2$max for the girls is 28.13ml.kg.min^{-1} and 49.14ml.kg.min^{-1}, respectively and the 23.7ml.kg.min^{-1} is seen as an outlier. The median for the girls is 38.11ml.kg.min^{-1}. The 25th and 75th percentiles for the girls are 34.78ml.kg.min^{-1} and 41.43ml.kg.min^{-1}. Percentage body fat was identified as one of the determents of $\dot{V}O_2$max for boys. $\dot{V}O_2$max for boys is grouped in those with a percentage body fat higher than 35.15% and those with a percentage body fat lower than 35.25%. The range for $\dot{V}O_2$max for boys with a percentage body fat lower than 35.15% is 40.77ml.kg.min^{-1} to 76.05ml.kg.min^{-1}. The median for this group is 58.41ml.kg.min^{-1}. The 25th and 75th percentiles of this group are 53.37ml.kg.min^{-1} and 63.54ml.kg.min^{-1}. The range for $\dot{V}O_2$max for boys with a percentage body fat higher than 35.15% is 20.61ml.kg.min^{-1} to 55.89ml.kg.min^{-1}. The median for this group is 45.81ml.kg.min^{-1}. The 25th and 75th percentiles for this group are 34.78ml.kg.min^{-1} and 50.85ml.kg.min^{-1}.

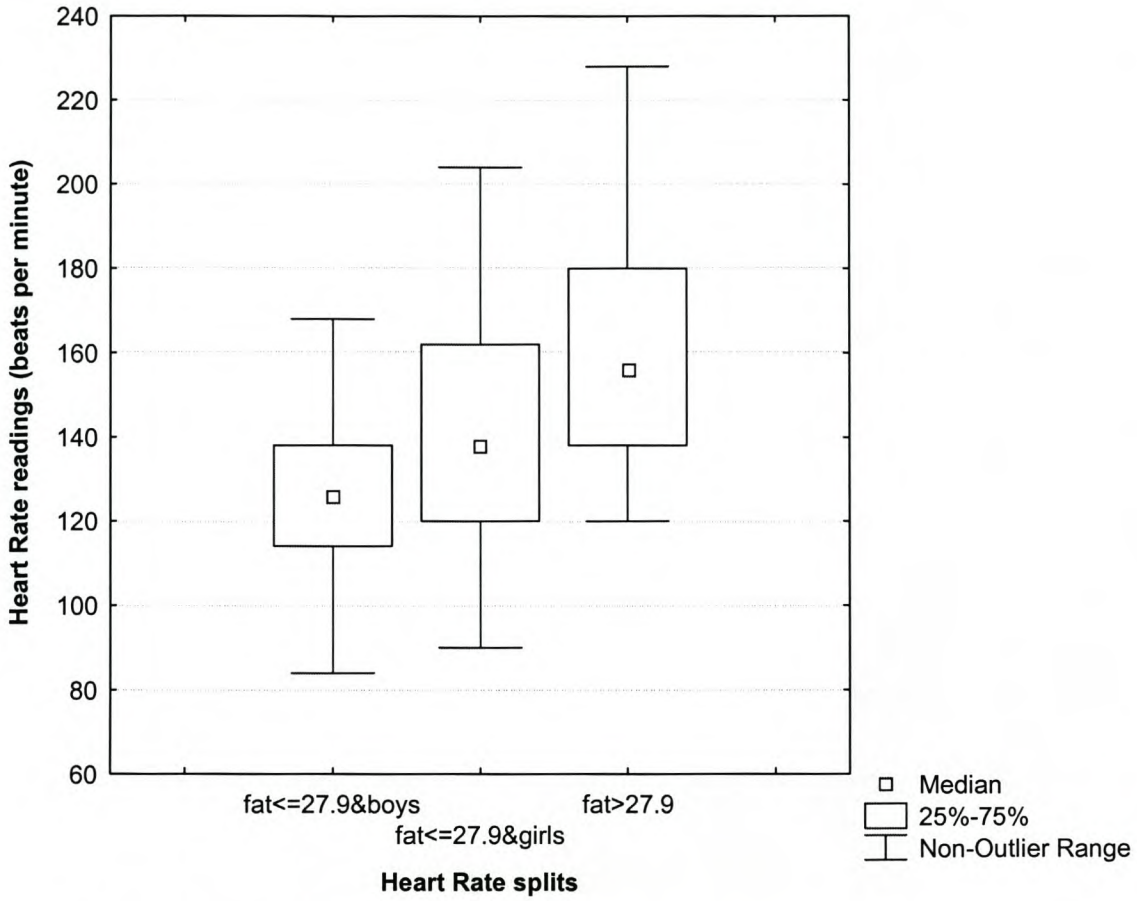

Figure E.5 A box plot illustrating heart rate splits.

Heart rate for boys with a percentage body fat lower or equal to 27.9% ranged from 84 to 164 beats per minute. The median for this group was 128 beats per minute. The 25th and 75th percentiles for this group were 116 and 138 beats per minute. Heart rate for girls with a percentage body fat lower or equal to 27.9% ranged from 90 to 204 beats per minute. The median for this group was 140 beats per minute. The 25th and 75th percentiles for this group were 120 and 162 beats per minute. Heart rate for subjects with a percentage body fat higher than 27.9% ranged from 120 to 228 beats per minute. The median for this group was 158 beats per minute. The 25th and 75th percentiles for this group were 158 and 180 beats per minute.

Appendix F

Summary of regression tree methodology

In the case of regression trees the dependent (response) variable y is a continuous variable. (For categorical response variables, a similar technique called classification trees can be used.)

1. Case 1: One continuous independent variable (*x*)

The method selects a point x_s between the minimum and maximum of x that splits the data into two sets (or nodes in a tree). All the cases for which $x \leq x_p$ goes to the left node and all the cases where $x > x_p$ goes to the right node.

The point where the split is made is the point that most successfully separates the high response values from the low ones.

The procedure above is then repeated for each of the two nodes. Thus a binary split is made on each node using the criteria mentioned above.

Stopping rules are used to decide when the splitting process should stop. For example a minimum number of cases per node can be specified, and if that minimum number is reached, the node will split no further.

2. Case 2: One categorical independent variable

In the case of a categorical independent variable, all combinations of binary splits of the levels of the variable are considered and the combination that most successfully separates the high response values from the low ones are used as splitting criteria. For example if a variable has three levels namely *a*,*b* and *c* then the following combinations of splits will be considered:

Left node	Right node
a	*b*,*c*
a,*b*	*c*
a,*c*	*b*

3. Case 3: More than one independent variable (combination of continuous and discrete)

The procedure described above is applied to each variable independently. Then the variables are compared with one another and the one that provides the best split over all the variables is used as the splitting variable.

4. Variable importance

A variable importance factor in terms of its effect on the response variable can be derived once the tree has been built. This variable importance is calculated based on the number of times the variable was used as splitting variable and how well it separated the low values from the high values.

Appendix G

Formulas used

1. Formula used to calculate BMI

$$\text{BMI} = \frac{weight(kg)}{stature(m)^2}$$

2. Formula to calculate children's percentage body fat (Slaughter *et al.*, 1988)

% body fat (girls) = 1.33(tricep + subscapular) – 0.013(tricep + subscapular)^2 – 2.5

% body fat (boys) = 1.21 x (tricep + subscapular) – 0.008 x (tricep + subscapular)^2 x 1.7

3. Formula to calculate VO_2max (Appicelli, 1998)

$\dot{V}O_2$max (girls) = 65.81 – (0.1847 x step test pulse rate, beats per minute)

$\dot{V}O_2$max (boys) = 111.33 – (0.42 x step test pulse rate, beats per minute)